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THE REGULATION OF BODY TEMPERATURE.[1]

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THE physiology of body temperature regulation has aroused much interest in recent years, because the war compelled man to prepare himself for exposure to the cold of arctic regions and high altitude and to the heat of deserts, of tropical seas and of severely taxing industrial conditions. Physiologists both here and abroad have been interested in determining the nature and limitations of man's ability to compensate for such environmental hazards.

We all know that the majority of animals are poikilothermic—their body temperature rises and falls with that of their environment. No animal obtains its body heat entirely from the environment, however, because living tissues elaborate some heat. Many forms, even invertebrates such as the little sand crab of our beaches, can compensate slightly for a lowered temperature; but these forms are unable to resist the major swings of temperature.[(1)] Man and other mammals and birds are warm-blooded and homeothermic. In these last-mentioned forms serious consequences result from a variation of only a few degrees from the normal mean temperature.

Before considering the elaborate mechanisms involved in body temperature regulation and their control, however, it will be well to point out for emphasis three physiological generalizations which such a study will reveal.

1. The constancy of body temperature is an excellent example of homeostasis. In 1878 Claude Bernard, the famour French physiologist, pointed out that, though a changing environment continually impinges upon a living animal, the cells which comprise the body really live in a relatively constant environment. *"La fixité du milieu intérieur est la condition de la vie libre."* The constancy of the "internal matrix" is essential to the functional existence of most organs of the body. The term "homeostasis" is that of Walter B. Cannon, of Harvard. In his book "The Wisdom of the Body" he has described the mechanisms which maintain the homeostatic state of the blood sugars, blood pH, salt balance *et cetera*. As one considers the mechanisms which operate in the regulation of body temperature, a clear picture is obtained of the elaborateness of peripheral adjustments and the delicacy of central control, essential to the maintenance of these constant states, which renders birds and mammals largely independent of many fluctuating conditions of their environmental temperature.

2. The endocrine system and the nervous system are both involved in the regulation of temperature as they are in the maintenance of water balance. Furthermore, there is a cooperation between the two systems which must be mediated by some mechanism which is still not clearly understood. I will endeavour to tell you what is known about it.

3. The control normally exercised by the central nervous system is not all vested in one nucleus or portion of the brain or spinal cord. Textbooks of physiology state that the nervous system is roughly organized on the basis of "levels of function".

Primitive forms possess neither the complex nervous systems nor the refinements of reactions and control which characterize birds, mammals and man. The generally accepted concept is that, as higher centres developed within the nervous system, they added new functions and took over control of the primitive simple reactions. It was pointed out by Hughlings Jackson in 1884 that when these higher levels of integration are destroyed by disease, or removed surgically, two distinct groups of changes occur: deficiency phenomena and release phenomena.

This developmental viewpoint emphasizes the phylogenesis and ontogenesis of temperature regulation. Many of the primitive Australian animals present an excellent

[1] A post-graduate lecture delivered under the auspices of the Australian Post-Graduate Federation in Medicine.

example of a poorly developed regulatory mechanism.[25] They cannot maintain a constant body temperature as well as can higher forms, and they show a greater rebound on modification of heat production. With phylogenetic development comes a more highly developed control and a greater freedom of action.

Young birds, young mammals and babies have a rather poor temperature control. Baby chicks do not shiver until the seventh day after being hatched. The temperatures of children are peculiarly labile.[26] The delicacy of higher control develops with maturation of the nervous system.

This concept of the cephalization of control within the nervous system can be well illustrated by the studies I am about to review. Body temperature is regulated by activity in more than one region of the central nervous system. Higher centres contribute refinement of response which lower centres are incapable of executing.

With these three points to be illustrated—(i) maintenance of homeostasis, (ii) neuro-endocrine cooperation, and (iii) the evolution or "evolvement" of various interdependent centres of integration within the central nervous system—we will proceed with an analysis of temperature regulation.

The Norm and Normal Variation of Temperature.

The thermostat in man is set at 98·36° F. or 36·87° C. It is rather interesting that all completely homeothermic mammals have a temperature set at practically the same level. Birds are stabilized at a higher temperature level (41·8° C.). It is this higher level of temperature, activity and metabolism which must make birds more sensitive to oxygen deprivation and respiratory poisons such as carbon monoxide, and thus useful to miners. Large birds have lower temperatures than the smaller species with larger surface areas. The temperature of an ostrich is not greatly different from that of man. Temperature in birds is regulated in very much the same fashion as in mammals.

One does not expect a thermostat to hold temperature exactly on a line. How delicate is man's thermostat, and what are the speeds of the various opposed responses which maintain the homeostasis?

Diurnal variations in body temperature occur. They are not present at birth, but are fully established in man by the end of the second year. The cycle disappears during a long interval of unconsciousness—in sleeping sickness. Curves show considerable variation. There are "morning" individuals who get up early, have a morning peak, and then the temperature falls and they go to bed early. An "evening" individual gets up late, is sluggish all morning and really does not "get going" until evening—his peak comes then.[16] One's efficiency moves with his temperature. If you are a "morning" type, do not gamble or do business with an "evening" individual at night—drag him out of bed in the morning.

Most individuals reach a maximum temperature between 4 and 5 p.m. according to older books and between 5 and 7 p.m. according to newer texts—is life changing? There is a corresponding low point in the early morning hours (5 to 6 a.m.). This diurnal swing is not much more than 1° C. The peaks are not completely reversed in night workers, but they are in nocturnal animals. The fluctuation appears to be a phenomenon associated with activity cycles. The flattening of the curve and incomplete reversal in men on night shifts probably merely indicate less rest during the day sleeping and less work during night working[6] (Figure I, A).

There is likewise a monthly temperature cycle, and claims have been made that the time of ovulation can be detected by sudden changes in the body temperature level. Unfortunately some gynæcologists claim that ovulation occurs when there is a high mid-cycle temperature followed by a drop, while others claim that ovulation occurs when a low temperature is followed by a sharp rise[15][28] (Figure I, B). Disregarding possible interests in whether or not pregnancy can be ensured or avoided on the basis of information obtained with a thermometer, it can be said that there is a cycle, but the changes in temperature are quite variable. This gives us some indication of the over-all tolerance of the thermostats regulating body temperature.

The high premenstrual temperature in the uterus could certainly be explained on the basis of blood flow and proliferation. The hormones producing this might affect the blood flow to other membranes in a similar fashion—there are reasons for thinking so. The fall in temperature during catamenia could likewise be explained on a mechanical vascular change such as Markee's work on blood vessel constriction in menstruation would show. The mid-cycle peak in temperature associated with follicular development could be explained on the basis of activity changes. In animals in which activity has been studied there is a peak of activity at the time of ovulation. Œstrogen injections produce augmented activity. Whatever its causes may be, the variation amounts to very little more than the daily change—1·5° C.

The Opposing Arms of the Control Mechanisms.

An animal can meet an environmental change and maintain its homeostatic condition by (i) producing and conserving more heat if exposed to cold, or by (ii) diminishing heat production and augmenting heat loss if exposed to heat. To maintain a normal body temperature there normally must be a balance maintained between heat intake *plus* production on one side and heat loss on the other.

The physiological processes comprising the two sides of this balancing mechanism are well shown in the following tabulation, modified from Du Bois.[10]

FACTORS INCREASING HEAT PRODUCTION AND LOSS BY THE BODY.

A.	B.
Heat Contributed to Body.	*Heat Lost from the Body.*
Carbohydrate.	Vaporization.
Fat.	Radiation.
Protein.	Convection.
Exercise.	Panting.
Shivering.	Sweating.
Unconscious tension of muscles.	Insensible perspiration.
Increased basal rate—	Artificially increased surface moisture.
1. Disease.	Increased surface exposure.
2. Endocrines.	Changed temperature gradient (cooler environment).
Specific dynamic action of foods.	Increased skin circulation.
Applied heat—	Increased air movement.
1. Food.	Basal heat loss (metabolism).
2. Sun.	Decreased contribution by factors under A.
3. Heating systems.	
Condensation of vapour.	
Basal heat production.	
Decrease of heat dissipation by factors under B.	

Hypothermia, 35° C.; normal, 37° C.; fever, 39° C.

A fuller knowledge of the nature, effectiveness and control of the activity of these factors can be obtained from a consideration of the responses of animals when exposed to heat and cold. But first I think it might be well to consider the afferent side of temperature regulation. How does the body know it is too hot or too cold, and how is the intensity of responses rendered appropriate to the requirements of the moment?

The Afferent Side of Temperature Regulation.

Sense Organs.

Specialized sensory organs sensitive to heat and the absence of heat (cold) are present in the skin, as we all know. The cold-sensitive endings are presumably the Krause end bulbs, which lie superficial to the arterial net and respond to the gradient of temperature between the surface and the vascular layer.[3] One suggestion as to how they operate is that a quick change of temperature on the surface steepens the gradient and the ending is subject to deformation. If cold exposure lasts for a long time and the entire layer of skin cools, the gradient flattens and the deformation lessens—consequently the ending fires less frequently. That is why reflex responses to cold are generally of short duration. The threshold

for cold is a fall in temperature at the rate of 0·004° C. per second. An external temperature of 20° C. or below is necessary to give this gradient. This gradient must hold for three seconds to stimulate. Bernhard and Grant (*The Journal of General Physiology*, Volume XXIX, 1946, page 257) believe that other mechanisms operate in firing these sensory receptors.

Warmth-sensitive endings (Ruffini endings) require a use of 0·001° C. per second for at least three seconds to fire them. These endings adapt quickly, and that is the reason why reflex responses to heat likewise tend to be of short duration. External temperatures must be 45° C. or over to give the required heat gradient.

The responsiveness of an animal to any thermal stimulus depends upon an area factor, upon an intensity factor, and upon the previous stimulation history—one is much more aware of cold on coming from a warm environment than when coming from cool surroundings. These endings respond to body surface temperature. The

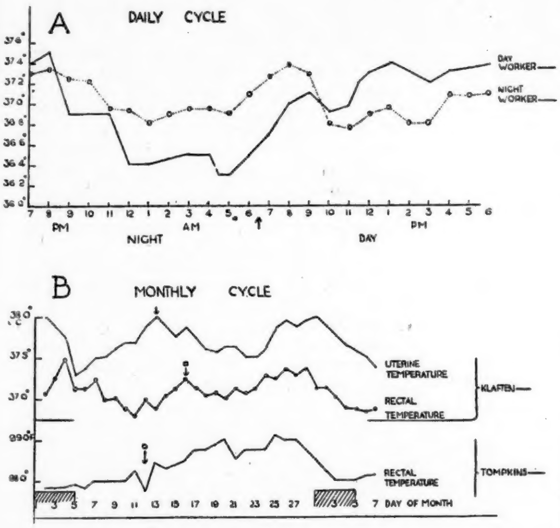

FIGURE I.

The range of normal body temperature variations as shown by (A), the diurnal temperature cycle and the incomplete reversal during periods of night work, and (B) the monthly temperature variation associated with cyclical activity of the reproductive glands.

internal temperature may be quite normal; but if the skin is chilled one is induced to shiver so violently that rectal temperature may even rise. It is likewise possible to play tricks on the body of the reverse kind. If on a cool day one shines a warm light on a dog's skin he will pant, and thus may even drive his internal temperature so low that he will have quite a chill. A dog may pant while he shivers. Leonard Hill many years ago found that if a man sitting in a hot room perspiring places his hand in ice water he ceases to perspire, and a rise in rectal temperature, a fever, results.

Severing the nerves from these endings or transection of the well-known spinal pathways involved abolishes the reflex control but does not render animals poikilothermic.

The Blood Temperature.

The body's second line of defence is a sensitivity of brain centres to blood temperature. Heating or cooling of the carotid blood, heating or cooling of the brain directly, will evoke a response. The skin-induced reflexes are supported by a direct activation of the centres by the warm or cold blood as soon as it reaches the brain from the skin. A change of 0·2° to 0·5° C. evokes a response. More will be said concerning the locus of these centres later. It has been demonstrated many times that changes in blood temperature caused by drinking a deal of hot fluid or cold ice water will evoke reactions, even though skin temperatures are kept constant.

The importance of blood and surface temperatures varies in different animals. Skin temperatures in the chick or cat will not produce a response in the absence of a blood temperature change. Dogs are very sensitive to skin temperature changes (Figure II).

Compensation against Exposure to Cold.

The problem which presents itself is, how does and how can man protect himself against brief exposure to temperatures of 0° up to 20° F. and down to –40° F. and avoid frost-bite and death. Or, as in the case of visitors to New Zealand and southern Australia, how can he protect himself against constant exposure to temperatures of 50° to 60° F. and avoid chilblains and erythrocyanosis of the legs.

The compensatory reactions observed to occur in animals on exposure to cold fall in different categories: some are immediate and some appear slowly; some are of temporary

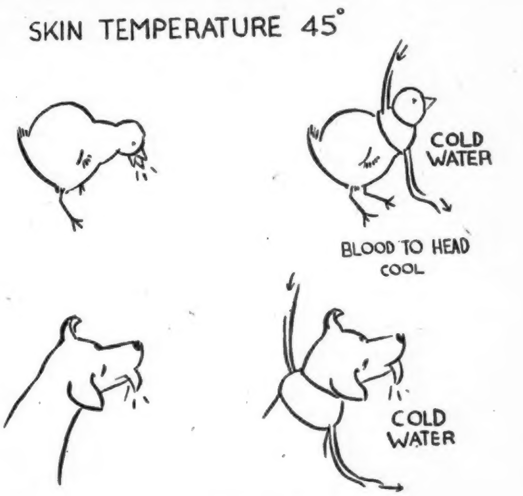

FIGURE II.

Showing the relative importance of surface temperatures in chick and dog. Cooling blood to the head abolishes panting in the chick but not in the dog.

duration and others are permanent; some are of endocrine or chemical origin, while others are neural. Tolerance of extremes depends upon the ability of these reactions to strike a balance between heat production and heat loss. The intensity of the reaction, of course, depends on the exposure. A temperature of 86° F. is the temperature of thermal neutrality of an unclothed resting individual—the temperature at which heat is neither lost nor gained by the body. For clothed individuals it is naturally much lower—how much depending on the clothing. When the body begins to lose heat, reactions soon follow which tend to balance the loss.

Piloerection—erection of fur or feathers—begins as soon as the skin is cooled below normal. The gooseflesh reaction is the remnant of this response in man. Piloerection is mediated by the sympathetic division of the autonomic nervous system, and occurs simultaneously with vasomotor changes. Heat loss by irradiation, convection and evaporation is cut down by the insulating layer of entrapped air. Man accomplishes the same thing by donning more clothing.

Vasomotor changes occur. Between 73° and 88° F. (23° and 31° C.) environmental temperature, an unclothed subject is comfortable because no change in body temperature occurs. Since the lower ranges are below thermal

neutrality, some compensation has occurred. Blood flow changes in the skin occur below 86° F., and they are sufficient to prevent body heat imbalance. Temperatures below 73° F. are regarded as cold.

Vasoconstriction of skin vessels begins immediately on exposure to cold. According to Sir Thomas Lewis[2] this closure is executed by the following three cooperating reactions: (i) there is a local constriction of skin vessels in the cold; (ii) there is an immediate but transient general vasoconstriction of peripheral sensory origin; (iii) this transient reflex is supported and supplemented as soon as blood cooled at the body surface reaches the brain.

When maximal vasoconstriction does occur, it involves arteries, arterioles, capillaries, venules, and even veins of the skin. An arm, for example, becomes pale, cold and practically pulseless. In order to preserve life, the organism, by these vasomotor reflexes, conserves internal heat by sacrificing surface structures and extremities. Little effort is made to maintain skin temperatures. They drop with the environment.[1] In this way an insulating shell of three to five centimetres in thickness is provided. The area from which heat may be lost is cut down by 25% by this sacrifice of the extremities. The effectiveness of this conducting layer as an insulator may not be so very great, however, because the conductance of muscle and skin is close to that of water. Fat tissue, however, has only half the conductance of muscle and will maintain (per given thickness of layer) twice the temperature gradient. The female's predilection for a subcutaneous fat layer, in addition to adding to her charm, enables her to endure immersion in cold water for a longer time than men. The fact that high fat diets confer greater immunity to cold than equicaloric diets of carbohydrate or protein is ascribed (Glickman, Keeton *et alii*[3]) to the fact that much fat is immediately moved to and stored in subcutaneous tissues. Eating frequently is a help in this direction.

The superficial layers are not sacrificed, however, without a struggle. Lewis has described a reaction tending to do this.[2] When a hand is immersed in a mixture of ice and water the skin temperature of the fingers drops to 0° C. Within five to twenty minutes, however, the fingers suddenly begin a feel a little warmer. Actual measurements show a flushing and a temperature rise of as much as 8° C. These vasodilatations occur periodically and last for brief intervals; they occur in all extremities—ears, nose and face. According to Lewis they are due to axone reflexes initiated by mild degrees of tissue injury. The reflex begins to operate at 15° C. (59° F.). It is this type of injury which, if permitted to proceed for long (two hours) will produce chilblains, erythrocyanosis of the legs, trench foot, "immersion hand". The reaction does tend to prevent frost-bite and slow down the process of injury.

At an external temperature of 75° F. the unclothed body of some individuals can maintain a gradient between skin and internal temperature of 10° F. Under these conditions 70 Calories per hour of heat are lost. This is about equivalent to basal heat production of the body (56 Calories per hour per square metre—man's surface area is about 1·5 square metres). If the heat loss is much greater, then other reactions must be brought in to supplement heat production, because by inhibiting sweating, erecting dermal appendages and depleting the skin of blood, an animal has made the maximum adjustment to cut down heat loss. He can do a little more by curling up and cutting down surface exposure; but this, too, has its limitations (25% reduction). At 30° F. the temperature gradient is 30° or more between the skin and the interior, and the heat loss is as much as 700 Calories per hour.[1]

There are four general ways in which the heat production of the body can be augmented.

The adrenal medulla is involved. Cannon and his associates showed that the same stimuli which evoke sympathetically mediated piloerection and peripheral vasoconstriction produce simultaneous discharge of the adrenal medulla. The adrenal medulla is one of the three endocrines of nerve origin. It is the counter part of the post-ganglionic sympathetic neurone and elaborates a hormone which has the same effect as the chemical mediators elaborated by these neurones. Adrenaline reinforces the sympathetic; it liberates sugar from storage and it increases the basal metabolic rate of heat production. This is an endocrine factor in temperature regulation. Some fever-producing toxins stimulate the adrenal medulla.

Muscular activity augments heat production. The maximum energy output on a bicycle ergometer is about 490 Calories per hour, and of these, 402 Calories per hour can go into heat.[1] A naked man cannot meet the 700 Calories per hour lost at 30° F., however, and he can work at this maximum rate for only 354 hours.

Reflex shivering induced by a cold skin or shivering induced by cold blood is the chief method of augmenting heat production. When exposed to a temperature of 55° F., a naked person begins to shiver within ten minutes. In colder air shivering begins immediately, and it may be sufficiently intense to enable a person to maintain his body temperature for one hour when immersed in water at 0° C. (32° F.). It begins as single muscle unit activity of some five twitches per second and grows to twelve per second; then grosser twitches of muscle elements begin, which gradually involve practically all the muscle groups, even of the hands and face, in asynchronous contractions.[7] The maximum heat produced by shivering is 370 Calories per hour.

Thyreoid compensation takes place. Within seven to fourteen days after exposure to cold the thyreoid gland hypertrophies and begins to secrete more thyroxine or metabolism-stimulating substance.[18][19] This raises basal heat production by 20% to 40%, it is claimed. The thyreoid is purported to become more active because of a greater liberation of thyreotropic hormone from the anterior lobe of the hypophysis due to influences exerted on it by the temperature-regulating centres of the hypothalamus. If the neural connexions between the hypothalamus and the pituitary gland are severed, the thyreoid does not become more active on exposure to the cold.

Exposure of the unclothed body surface to sunshine in winter at 30° F. is the equivalent of a temperature rise of 11° F. and saves 90 Calories per hour. If wind velocity is quadrupled, heat loss by convection is augmented by a similar amount (quadrupled).[1]

In the cold there is a cold diuresis of 300 to 450 millilitres per hour for the first two hours or so. There are a rise in blood pressure and a concentration of the blood. All these last-mentioned phenomena are probably due to the peripheral shut-down of blood vessels and the confinement of the blood to a smaller capacity.[3][4]

The body cannot produce enough heat to meet the 700 Calories per hour drain of naked men exposed to 30° F. To protect himself in the colder environments man must wear clothes; but this does not solve all problems of existence at high altitude and in the polar regions.

Cherry-Gerard, of Scott's polar expedition (1922), made the following statement:

> On the most bitter days—it seemed that we must be sweating and all this sweat, instead of passing away through the porous wool of our clothing and drying off us, froze and accumulated. It passed just away from our flesh and then became ice—but when we got into our sleeping bags, if we were fortunate, we became warm enough during the night to thaw this ice: part remained in our clothes, part passed into the skins of our sleeping bags, and soon both were sheets of armour plate.

Much of this water loss from the skin is insensible perspiration; but in order that one may live, when quiet, clothing must be so heavy that in activity the body surface overheats and sweating results. In recent army tests it has been shown that a man (clothed for the Arctic), walking at the rate of 3·5 miles per hour at a 6·5% grade, produces 500 grammes of sweat in one hour. Of this, 400 grammes remain in the clothing.[6]

This condensation in the clothing has two serious effects. Firstly, it cuts down the efficiency of cooling by

evaporation. The principle of sweat cooling is that heat from the body is lost by vaporization of the water of sweat and the body surface is cooled. When this vapour condenses in the clothing again, however, the process is reversed and heat is redeposited in the clothing. In this way the cooling efficiency of the sweating is cut down by 25% to 60%. To eliminate heat, therefore, the body must sweat more. The second serious effect is that described in part by Cherry-Gerard. Ice is formed which not only destroys the insulating "warming properties" of clothing, but acts as a mechanical obstacle. The only solution to this yet found is to remove and dry the outer garments at intervals. Even the best clothing alone cannot protect an inactive man when he is fully exposed to –20° or –40° F. An injured man will die within four hours unless he reaches shelter or warmth.

At rectal temperature of 95° F. heat production begins progressively to diminish. At 75° F. shivering stops. Man can recover from a rectal temperature of 75° F. if handled properly.[27] Hibernating animals will fall to 32° F. and recover, and cats and dogs have been brought back from 50° F. One of the large items in collapse at low temperature is just ordinary fatigue. A person collapses if he works maximally for four hours, and he does the same if he shivers maximally for that period. He becomes fatigued, he exhausts the available fuels of muscular exercise, his muscles begin to contract poorly, he becomes cold, and at a time when survival depends upon activity and wakefulness he becomes sleepy. Cold is like a depressing narcotic; the individual becomes sleepy and paralysed and then dies. The only physiological acclimatization to cold is that of the thyreoid. There is also a psychic factor, but this lies outside our realm.[1]

Exposure to Heat.

I am not going to elaborate the story of protection against overheating. Each of you is probably an expert at that here in Australia, and in the laboratories of your medical schools much work in this field has been done.[20] There are likewise many good reviews of this subject.[9]

A few degrees' rise in body temperature are much more serious than a few degrees of fall. A few individuals have recovered from a temperature of 109° F., but this is the upper limit. Man's protective devices against heat are so very good, however, that he can endure for long periods of time temperatures of well above 100° F. and for brief periods of time temperatures which coagulate unprotected protoplasm within a few seconds (225° F. *et cetera*).

Heat loss is by radiation, convection (conduction) and vaporization. The emphasis placed on these individual pathways depends on environmental conditions. At low temperatures radiation and convection are more important than vaporization. For example, a nude subject in a basal state at 22·7° C.[1] loses heat in this ratio: 19% by vaporization, 66% by radiation, 15% by convection. This is reversed at high temperatures.

When exposed to heat, an animal has really only one method by which it augments heat loss. To be sure, there are a few vasodilator fibres; but the great amount of peripheral shunting of blood is due to relaxation of the vasoconstrictor tone and cessation of adrenaline discharge. Activity is reduced, and there is a diminution of thyreoid action which enables the basal metabolic rate to fall. The evaporation of fluids from mucous membrane surfaces in panting must bear the brunt of heat dissipation in hot environments. The body can lose a kilogram (2·2 pounds) per hour. As the temperature rises, there is some augmentation of insensible perspiration. From 88° to 98° F. sweating in individuals at rest begins. Since the evaporation of 1·0 millilitre of water requires 0·58 Calorie, evaporation of 1000 millilitres of sweat per hour dissipates 580 Calories. This figure is far above the basal heat production and gives some idea of the protective margin of safety against overheating (eight times 70 Calories per hour). The maximal exposure to the sun contributes only 200 Calories per hour.[22]

On exposure to high temperatures a person is at first much more uncomfortable than he is after a day or two. His ability to work likewise increases as the acclimatization occurs. What is the nature of this adjustment? Acclimatization to heat is largely a matter of cardio-vascular adjustment.[4][9][13][14] The strain placed on the circulation by the excessive demands of the skin for an increased blood supply to serve the requirement of heat regulation is very large. It has been estimated by Adolph (*The American Journal of Physiology*, Volume CXXIII, 1938, page 486) that approximately 2·5 litres of blood per minute must be circulated through the skin to dissipate the heat production of walking in the desert. In the unacclimatized man this increased circulation through the skin produces the early signs of peripheral circulatory collapse—that is, a rapid weak pulse, a decreased stroke volume and in some cases a decreased minute output, a large increase in pulse rate and a drop in blood pressure on assuming the upright position (table tilting or just standing up). The vasomotor system is unable to keep the great veins filled.

The acclimatization or cardio-vascular adjustments are largely made in the first four days (Compton test—table tilting). Heart rate and blood pressure and rectal temperature changes on working in hot environments are reduced in four to eight days to a steady state; if not, the individual cannot tolerate that environment. These improvements are brought about in three ways: (i) There are a dilution of the blood and an increase in volume to fill the greater capacity. (ii) There is an increased sensitivity of the vasomotor apparatus of the autonomic nervous system. This is shown by the cold pressor test (cardio-vascular response to placing left hand in ice water for sixty seconds), which gives a greater response. (iii) There is a gradual augmentation of peak sweating, as though the heat dissipation centre had become more responsive. Strangely enough individuals in the tropics are reported to have more sweat glands than those in non-tropical areas.[17]

When once this acclimatization develops, it lasts (though diminishing) for four to eight weeks, even though the individual is returned to a cold environment.[13] As acclimatization represents a sensitization of the sweating, panting and vasodilator centres, fevers seem to represent a desensitization of these centres and a stimulation or sensitization of centres for heat production and conservation. Fever-producing drugs and toxins cause vasoconstriction, adrenaline discharge and shivering. The temperature goes up and up, away beyond a level which would normally produce sweating and profound vasodilatation, before these centres break through the inhibition. It would be very interesting to test directly by modern electro-techniques the sensitivities of the centres controlling temperature regulation under the vaious conditions mentioned. It should be possible, because we now know approximately where these centres are located.

Centres Involved in the Regulation of Body Temperature.

Information concerning the existence and locus of centres which control the temperature-regulating reactions has been obtained by stimulation or by injury to or ablation of various parts of the central nervous system.

The Isolated Spinal Cord.

Despite Foerster's claims that the spinal grey matter is sensitive to blood temperature changes, it is now certain that the observations of Gordon Holmes (1915) on spinal man and of Sherrington (1924) on spinal animals are correct—that the spinal cord does not contain heat-regulating centres. Spinal animals and man are practically poikilothermic.

As recovery from spinal shock occurs, more reflex activity is possible and a greater average heat production develops.[23] Blood pressure is reestablished and a degree of vasomotor responsiveness reappears, but never effective temperature control. Sweating occurs as part of the mass reflex; heat does elicit reflex sweating in some species, but sweating is likely to be inappropriate. Shivering does not occur. The sympathico-adrenal system can be activated

reflexly by nociceptive stimuli but not by cold. Peripheral vessels tend to remain dilated; piloerection cannot occur; fever-producing drugs and toxins have little effect on body temperature.

The two compensatory mechanisms remaining intact after cervical transection of the cord are panting, in which the diaphragm above is employed, and thyreoid hyperactivity induced by cold. This latter change *plus* the general raised activity of cardio-vascular and muscle tone can account for the slightly greater resistance to cold which spinal animals gradually develop during the first two to three weeks after operation.

The Decerebrate Animal Medulla and Cord.

A few chronic decerebrate cats and dogs have been prepared[24] and a few human cases have been known in which lesions practically severed the brain stem at the mesencephalic level. These likewise are poikilothermic and must be constantly protected against the normal fluctuations of environmental temperature if they are to survive. They do, however, possess greater abilities than do spinal animals. Decerebrate cats and dogs have the entire quota of vasomotor reflexes and much motor ability. They eventually are able to stand and to walk. Very low blood temperatures induce imperfect shivering, and high blood temperatures produce low-grade panting. These are ineffective rudiments of the temperature-regulating reactions.

The Cerebral Cortex.

According to the theory of cephalization of control, the cerebral cortex should have added some refinement of function and assumed a degree of control over lower centres of temperature regulation.

Stimulation of the cerebral cortex does produce piloerection and vasoconstriction and will facilitate panting; but this does not necessarily indicate any rule in temperature regulation. Cerebral hæmorrhages inactivating the cortex in man may produce a failure of thermally induced skin vasoconstrictor and vasodilator reflexes. Temperature sense remains, however. Laboratory experiments on animals have shown that the cerebral cortex plays only a minor role in temperature regulation. After the cerebral hemispheres have been ablated, skin vessels remain chronically somewhat dilated; heat loss is thus greater. Animals permit their body temperature to fall abnormally before beginning to shiver, and then they over-shiver. The same is true of panting. They appear to have become less responsive to skin temperature stimuli, and show the less delicate responses to blood temperature changes. The cerebral cortical centres thus contribute or are essential to a refinement of body temperature regulation.

The Corpus Striatum and Thalamic Areas.

It was once thought that the *corpus striatum* and thalamus were involved in the integration of temperature-regulating responses. Many of the effects produced by stimulating and warming and cooling these structures were due to effects on adjacent hypothalamic areas.

The Hypothalamus.

The centres which integrate the autonomic, endocrine and somatic reactions that maintain body temperature homeostasis are located within the hypothalamus (Figure III).

The regulation of heat dissipation is dependent upon structures in the anterior preoptic and dorsal areas of this nuclear complex. Electrical stimulation of this anterior region has been reported to produce a generalized vasodilatation, a fall in blood pressure, sweating and panting. Stimulation of the preoptic region by means of heat produced by warm objects or by high-frequency currents causes all the phenomena which favour heat dissipation.[30]

Lesions produced in this area by tumours or by electrolytic coagulation of the nuclei abolish the reaction of panting, sweating and vasodilatation which result from warming the skin and the blood. The animals are sensitive to high temperatures; they cannot prevent a body temperature rise on exposure to heat. The antero-lateral and dorsal sections of the hypothalamus are essential to normality.

Regulation of heat production and heat conservation is carried on by the posterior lateral hypothalamic area. Stimulation of this general area produces vasoconstriction, adrenal medulla discharge and piloerection—activity of the sympathetic division of the autonomic complex. Application of cold to the area has given less satisfactory results, but it has been claimed that thermal stimuli (cold) will incite a response. Lesions in this region abolish shivering, impair vasomotor responses and render animals deficient in response to cold. Large lesions in this posterior portion of the hypothalamus tend to impinge upon the descending paths from the heat control centres; poikilothermic animals can thus be produced by large posteriorly placed lesions. The tuberal regions and hypophyseal stalk must be left intact if the thyreoid reaction to cold is to occur.

In man, hyperthermia, hypothermia and a degree of poikilothermia develop as a result of tumours and lesions involving the hypothalamus. Such lesions are generally

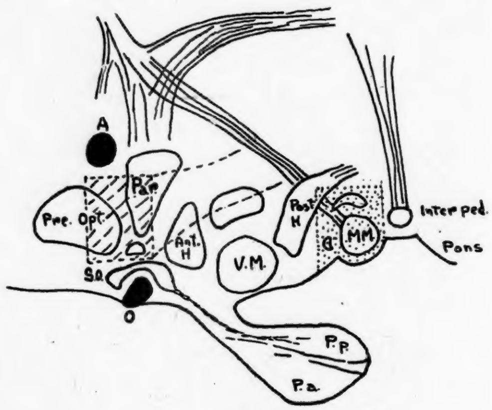

FIGURE III.
Diagram of the hypothalamus showing the areas essential to normal heat dissipation (cross-hatched) and the general area essential to normality of heat production and conservation (stippled).

large and far from confined to any one group of nuclei. According to surveys of Davison, Zimmerman and others,[2][5] however, hyperthermia is generally associated with rostrally located lesions and hypothermia with lateral and caudal hypothalamic injury, and in cases in which there is a rather complete absence of temperature-regulating ability practically the entire hypothalamus has been destroyed. The clinical literature does not contradict the conclusions of the laboratory, although, as one might expect, many cases are apparently somewhat difficult to fit into the picture.

Conclusion.

In conclusion, it can be stated that both the clinical and laboratory findings to date support the belief that the nuclei of the preoptic and superoptic regions of the hypothalamus regulate the sweating, panting and vasomotor responses favouring heat dissipation. The lateral and caudal regions of the hypothalamus contain those centres essential to the somatic and autonomic reactions which augment heat production and heat conservation. From these regions impulses or influences must pass to the hypophysis and the motor nerves which control the effector organs involved in the activities essential to temperature regulation. These are the centres which cooperate to maintain the constancy of body temperature essential to

the normal body function; but the exact roles played by the various individual nuclei are still unknown.

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THE DIAGNOSIS, PREVENTION AND TREATMENT OF PUERPERAL INFECTION.[1]

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AT the present day a great deal is known of the bacterial causes and clinical aspects of puerperal infection.[2] But much of this knowledge is still not generally appreciated or has not been applied in practice, with the result that some of the hoped-for improvements in prevention and control have not yet been achieved.

It is our belief that lack of proper integration between the bacteriological and clinical services devoted to midwifery has been one of the chief factors retarding progress. For full efficiency a midwifery service requires that standard of teamwork which in war-time medicine produced such rapid and significant advances.

It is the purpose of this paper to summarize our present knowledge of the bacteriological and clinical aspects of puerperal infection, and to discuss the diagnosis, prevention and treatment from the standpoint of an integrated bacteriological and clinical service.

DEFINITION OF PUERPERAL INFECTION.

Puerperal infection is a wound infection of the genital tract. In every puerperal woman the placental site is an open wound, and in addition there may be lacerations or incisions of the uterine body or cervix, of the vagina, the vulva or the perineum. If during labour or during the puerperium pathogenic bacteria gain entrance to any of these wounds, the patient faces the dangers of puerperal infection.

THE BACTERIA RESPONSIBLE FOR PUERPERAL INFECTION.

The bacteria associated with puerperal infections can be divided, on the basis of their importance, into three groups, and are so listed, as follows.

Group I: Anaerobic streptococci, *Streptococcus hæmolyticus* group A, *Staphylococcus pyogenes*.

Group II: Hæmolytic streptococci of groups other than A, *Bacterium coli*, non-sporing anaerobic bacilli, aerobic non-hæmolytic streptococci, diphtheroids, *Clostridium welchii*.

Group III: *Neisseria gonorrhœæ*, *Streptococcus pneumoniæ*.

The first group, comprising the anaerobic streptococci, *Streptococcus hæmolyticus* group A and *Staphylococcus pyogenes*, is by far the most important from the point of view of both the frequency and the severity of the infections produced. The bacteria in the second group are much less important, either because they are less common or because they produce a less serious type of infection.

Infections due to the gonococcus and to the pneumococcus occur so seldom that these bacteria have been placed at the bottom of the list.

Frequency of the Various Bacterial Types in Melbourne.

During the two and a half years prior to July, 1946, 1341 cases of puerperal infection were studied. This series included all grades of clinical severity from fatal cases to those in which there was only one isolated rise in

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[1] Read at a meeting of the Victorian Branch of the British Medical Association on August 7, 1946.

[2] We employ the term "puerperal infection" instead of "puerperal sepsis" because the former is capable of accurate definition. The term "puerperal sepsis" should be discarded.

temperature, but in which bacteriological evidence of a genital tract infection was forthcoming.

The bacteria regarded as primarily responsible for these patients' infections are given in Table I. We have listed as anaerobic streptococci those strains which in the initial culture produced readily visible growth under anaerobic conditions, but which after the same period of incubation failed to grow in similar media incubated aerobically. The designation "anaerobic staphylococcus" has not been used. Some of the strains included under the heading "anaerobic streptococci" resembled staphylococci morphologically; but they did not show differences in colony form comparable with those seen with aerobic streptococci and staphylococci. Microaerophilic streptococci are not listed in this table. Strains which we regard as conforming to this description have occurred together with other bacteria, but in this series we have observed no infection due primarily to such a strain. Since many of the strains of anaerobic streptococci were not retested after several subcultures for their capacity to grow aerobically, it is probable that this group includes a small number of microaerophilic strains.

Under the heading "anaerobic Gram-negative bacilli" we have included all the non-spore-forming anaerobic Gram-negative bacilli cultivated from the genital tract. Many such strains show the characteristics of *Bacterium melaninogenicum*, but other varieties undoubtedly occur.

The aerobic non-hæmolytic streptococci have not been classified, since in our experience these streptococci are comparatively unimportant in puerperal infections.

TABLE I.

The Bacteria Responsible for 1,341 Cases of Puerperal Infection.

Type of Bacteria.	Number of Cases.
Anaerobic streptococci:	
Alone	213 } 1,093
With other bacteria	880 }
Streptococcus hæmolyticus group A	64
Aerobic non-hæmolytic streptococci	43
Bacterium coli	30
Staphylococcus pyogenes	26
Hæmolytic streptococci of groups other than A	20
Non-sporing anaerobic Gram-negative bacilli	12
Clostridium welchii	3
Neisseria gonorrhϾ	1
Unknown (no pathogenic bacteria detected)	49

In this series of 1341 cases anaerobic streptococci were held primarily responsible for 80% of the infections, although other bacteria (anaerobic Gram-negative bacilli, *Bacterium coli*, aerobic non-hæmolytic streptococci or diphtheroids) were usually present as well.

In comparison with infections due to the anaerobic bacteria, all other bacterial types of infection were uncommon. *Streptococcus hæmolyticus* group A was responsible for less than 5% of the infections and *Bacterium coli* and *Staphylococcus pyogenes* were responsible for slightly more and slightly less than 2% respectively. Only three infections due to *Clostridium welchii* occurred in this series, and not one of them was serious. There was only one gonococcal infection, and none due to the pneumococcus.

The figures in Table I must not be taken to represent the frequencies with which the various bacteria were recovered from the birth canal. In all types of infection, not only those due primarily to the anaerobic streptococci, it is common to isolate more than one type of organism. For instance, in 31 out of 64 cases of infection due to *Streptococcus hæmolyticus* group A other types of streptococci were also present. Similarly, although there were only 30 cases in which we regarded *Bacterium coli* as the important bacterial cause, this bacterium was often associated with anaerobic streptococci.

The predominant rôle of the anaerobic streptococci is further brought out in the results of blood cultures. In the five years prior to July, 1946, cultures were obtained from the blood of 53 patients suffering from clinical puerperal septicæmia.

Reference to Table II shows that anaerobic streptococci were recovered from 30 of these patients. Cases of septicæmia in the puerperium, in which the blood-stream invasion came from an extragenital source, have been excluded.

Sources of Infection.

On the basis of their source the bacteria responsible for puerperal infection fall into three categories: those which are introduced from without, those which are normally inhabitants of the vagina, and those which come from the viscera immediately adjacent to the vagina—namely, the bladder or the bowel. In Table III are shown the habitat and source of the bacteria most often associated with puerperal infections.

TABLE II.

The Bacteria Isolated from the Blood in 53 Cases of Puerperal Septicæmia.

Type of Bacteria.	Number of Cases.
Anaerobic streptococci:	
Alone	19 } 30
With other bacteria	11 }
Bacterium coli	6
Streptococcus hæmolyticus group A	5
Streptococcus hæmolyticus group B	2
Streptococcus hæmolyticus group C	1
Streptococcus hæmolyticus group D	1
Staphylococcus pyogenes	4
Aerobic non-hæmolytic streptococci	2
Diphtheroids	2

It will be seen that almost all the truly exogenous infections are caused by two bacteria only, *Streptococcus hæmolyticus* group A and *Staphylococcus pyogenes*. The bacteria in the second group, the hæmolytic streptococci of groups other than A, the anaerobic streptococci, the anaerobic Gram-negative bacilli, the diphtheroids and the aerobic non-hæmolytic streptococci, are fairly widespread in the human body, but in the great majority of cases their source in puerperal infection is the vagina. We are not aware of any experimental proof that in cases of infection due to anaerobic streptococci the source of the infecting organism has been the respiratory tract of the patient or those attending her. Nor is there proof that the bowel is the immediate source of infection. On the other hand, we have on record cases in which the strain of anaerobic streptococci associated with the patient's

TABLE III.

Sources of Infection.

Bacterium.	Habitat.	Source in Puerperal Infection.
		Exogenous.
Streptococcus hæmolyticus group A.	Upper part of respiratory tract.	Upper respiratory tract infections; wound and skin infections; healthy carriers.
Staphylococcus pyogenes.	Skin; upper part of respiratory tract.	Boils; carbuncles; wound and skin infections.
		Predominantly vaginal.
Hæmolytic streptococci of groups other than A.	Upper part of respiratory tract; vagina.	Vagina; occasionally throat in group C infections.
Anaerobic streptococci.	Vagina; bowel; upper part of respiratory tract.	Vagina.
Anaerobic Gram-negative bacilli, Diphtheroids, Aerobic non-hæmolytic streptococci	Upper part of respiratory tract; vagina; bowel.	Vagina; occasionally bowel in infections with aerobic non-hæmolytic streptococci.
		Predominantly Fæcal.
Bacterium coli.	Bowel.	Bowel; bladder.
Clostridium welchii.	Bowel.	Bowel.

infection in the puerperium was identical with that recovered from the genital tract before labour. The possibility of a urethral source in anaerobic streptococcal infections must also be considered.

In infections due to the typically fæcal bacteria, *Bacterium coli* and *Clostridium welchii*, the source and natural habitat are generally identical.

Danger of Spread, Method of Transmission and Severity of Infection.

Table IV illustrates the differences in the danger of spread, the method of transmission and the factors influencing severity in the different bacterial types of puerperal infection.

Puerperal infection can be sharply divided into the "infectious" and the "non-infectious", on the basis of the ability or otherwise of the causative bacteria to spread from case to case. Infections due to *Streptococcus hæmolyticus* group A and to *Staphylococcus pyogenes* are the only important bacterial types which are "infectious". In infection due to the other bacteria listed in Table IV the danger of spread of infection from one patient to another is negligible or absent. It cannot be too often emphasized that the anaerobic infections, which in our experience form the great majority of puerperal infections today, are not infectious.

Puerperal infection is transmitted by contact, by mechanical introduction, by droplets or by dust. In infection due to *Streptococcus hæmolyticus* group A, the very important part played by dust is still not fully realized. In staphylococcal infections, on the other hand, contact is the common method of transmission. Mechanical introduction is the important factor in infections with fæcal bacteria, although it should be remembered that in cases in which the urine contains *Bacterium coli*, infection of the genital tract may arise from this source.

Many of the factors influencing the severity of the infection in the individual case of puerperal infection are imperfectly understood. But in at least two types of infections, those due to *Streptococcus hæmolyticus* group A and to *Clostridium welchii* respectively, severity is due primarily to the virulence of the infecting strain (Butler and Hill,[1] Butler[2]).

Less is known of severity of the other types of infection. In infections due to anaerobic streptococci and to the other bacteria whose source is usually the birth canal, clinical experience shows that lowered maternal resistance, especially that occasioned by blood loss, is important. As yet it has been impossible to correlate the severity of the patient's infection with any particular attribute of the infecting strain. But work in progress in this hospital suggests that most of the infections which we now regard as due to anaerobic streptococci may be mixed infections. There is evidence of bacterial symbiosis in many instances, and in these it is possible that the infecting agent is not any particular bacterium, but rather a mixture of two or more bacterial types, and that the nature of this mixture of bacteria growing intimately together is a factor in determining the severity of infection.

Similarly, in cases in which the predominant bacterium in the infected genital tract is an anaerobic Gram-negative bacillus, an aerobic streptococcus other than *Streptococcus hæmolyticus* group A, or a diphtheroid, the severity of the disease is in part at least dependent on the associated bacteria.

Clinical Features of Puerperal Infection.

Puerperal infection may occur in any of the following grades of severity, each of which indicates a successive stage in anatomical spread: (i) infection localized to the birth canal; (ii) infection spreading beyond the birth canal, but localized to the pelvis; (iii) general peritonitis; (iv) septicæmia.

These grades are shown graphically in Figure I.

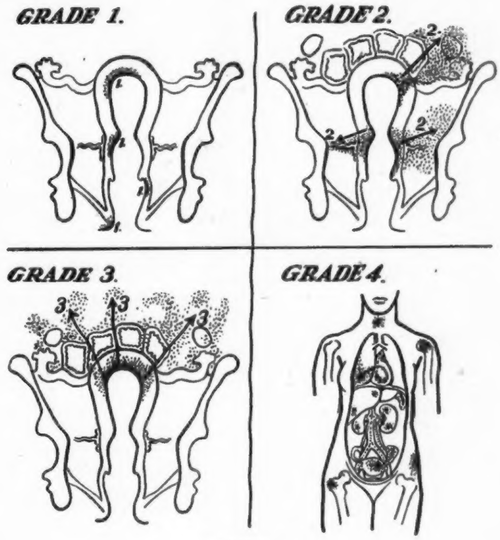

FIGURE I.

Infection Localized to the Birth Canal.

Infection localized to the birth canal is the mildest form of puerperal infection, and fortunately by far the commonest. The most frequent lesion is acute endometritis involving the placental site and decidua. Other lesions include infected lacerations or incisions of the uterus, the cervix, the vagina, the vulva or the perineum.

TABLE IV.
Danger of Spread, Method of Transmission and Severity of Infection.

Bacterium.	Danger of Spread.	Method of Transmission.	Factors Influencing Severity of Infection.
Streptococcus hæmolyticus group A.	Great and constant.	Droplets, dust, contact.	Virulence of infecting strain.
Staphylococcus pyogenes.	Present; greatest in babies.	Contact, dust.	Virulence of infecting strain; lowered maternal resistance.
Hæmolytic streptococci of groups other than A.	Negligible.	—	Unknown.
Anaerobic streptococci.	Nil.	—	Lowered maternal resistance (blood loss); tissue damage; probably bacterial symbiosis.
Anaerobic Gram-negative bacilli, diphtheroids, aerobic non-hæmolytic streptococci.	Nil.	—	Presence of other bacteria.
Bacterium coli.	Nil.	Mechanical introduction, infected urine.	Unknown.
Clostridium welchii.	Nil.	Mechanical introduction.	Virulence of infecting strain.

These patients are not seriously ill and their symptoms are few. Their rise of temperature and pulse rate is generally of a low order (Figure II), and the majority are afebrile within a week. Higher degrees of fever (temperature rise to 102° F. or more) and of tachycardia (pulse rate up to 120 per minute or higher) may occur as isolated phenomena in a benign course (Figure III), and very occasionally the illness maintains considerable acuity for a few days (Figure IV). It is rare, however, for fever even of a low order to persist for as long as ten to fourteen days.

Infection Spreading Beyond the Birth Canal but Localized to the Pelvis.

The three varieties of infection spreading beyond the birth canal but localized to the pelvis are pelvic peritonitis (including salpingo-oophoritis), pelvic cellulitis and pelvic thrombophlebitis.

With spread to the pelvis the patient's illness is more severe and more prolonged than when infection is confined to the birth canal. Her fever and pulse rate are of a higher order, and she generally shows, within a few days, such signs as lower abdominal discomfort or pain, difficulty of or pain on micturition or defæcation, and lower abdominal distension, tenderness and muscle resistance or rigidity. At the same time, or a little later, an ill-defined mass may be felt in one or other iliac fossa or through the vaginal fornices.

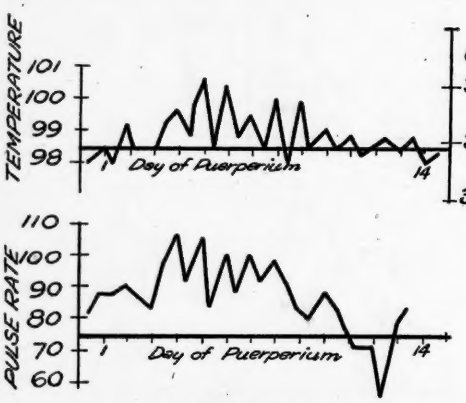

FIGURE II.

Pelvic cellulitis and pelvic peritonitis are not uncommonly present in combination, and in the early stages it may be impossible to define which is the predominant lesion. At a later date, however, the signs of differentiation become clearer. In general, inflammatory masses due to involvement of the pelvic peritoneum are palpable through the abdominal wall, are localized early and are discrete in outline. In contrast, the induration of pelvic cellulitis is best felt through the vaginal fornices, and extends, without clearly defined limits, from the cervix to the pelvic walls, producing, when the utero-sacral ligaments are involved, horseshoe constriction of the rectum. Pain is usually a more prominent feature of pelvic peritonitis than of pelvic cellulitis.

The diagnosis of pelvic thrombophlebitis must generally be one of exclusion, and *phlegmasia alba dolens* is evidence of its spread to the femoral vein.

General Peritonitis.

General peritonitis is a grave complication and is usually seen in the early days of the puerperium.

Its development is to be suspected when the patient with a raised temperature and pulse rate shows a fairly rapid deterioration in her general condition. At the onset of the peritonitis the temperature and pulse rate rise steeply, and although the temperature may fall after a brief interval, the pulse rate remains rapid or steadily quickens. As the infection progresses, the tongue quickly becomes dry, and the abdomen is increasingly distended.

In the established condition the abdominal distension usually extends to the flanks, and deep abdominal tenderness, and particularly "release tenderness", are almost invariably present; however, abdominal rigidity is uncommon and spontaneous pain a rarity. In the late stages appear rapid shallow respirations, muddy facies, persistent vomiting and peripheral circulatory failure.

General peritonitis may arise late in the puerperium from rupture of an abscess or separation of an adhesion in the pelvis. It is much less common at this time, and its onset is not usually accompanied by high fever, but by signs of profound collapse, with "running" pulse, subnormal temperature, falling blood pressure and peripheral circulatory failure. Figure VI is from a fatal case of general peritonitis due to anaerobic streptococci, and Figure VII is from a case of early general peritonitis due to hæmolytic streptococci of group A, in which a dramatic response followed the immediate institution of adequate sulphonamide therapy.

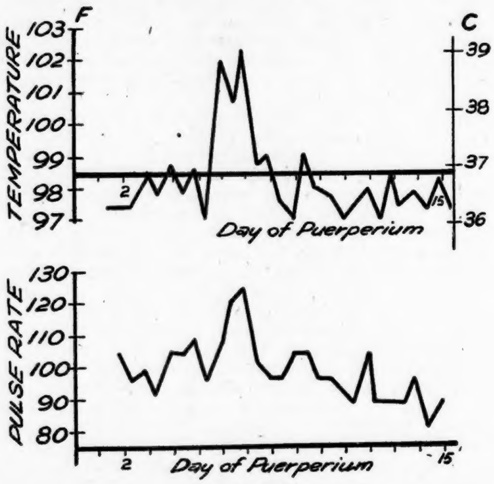

FIGURE III.

Septicæmia.

Septicæmia is the most serious grade of puerperal infection. It is that clinical state which results from the repeated or continuous inoculation of the blood stream with bacteria from a persistent focus, and in that it represents the repeated or continuous breakdown of bodily defences it is always grave.

Septicæmia is to be suspected in the presence of sustained or repeated high fever (temperature of 102° F. or higher) and a rapid pulse rate (120 per minute or over), particularly when these signs are not adequately explained by other lesions.

Figure VIII is from a fatal case of septicæmia due to anaerobic streptococci, and Figure IX is from a fatal case of septicæmia and infective endocarditis due to *Streptococcus hæmolyticus* group B.

CLINICAL ASPECTS OF CERTAIN BACTERIAL TYPES OF INFECTION.

Infections due to Anaerobic Streptococci.

Infections due to anaerobic streptococci are the commonest of all puerperal infections, of whatever grade of severity. They are aided by blood loss and tissue damage. The woman who suffers from severe anæmia of pregnancy, or from an exhausting labour, or from post-partum hæmorrhage, is a favourable subject for invasion by anaerobic streptococci.

A greater variety of clinical pictures may be seen in association with anaerobic streptococci than with any other bacterial type of puerperal infection. In the puerperium the common picture is nondescript, characterized by mild or moderate fever, a raised pulse rate and heavy or offensive lochia. With the severer grades of infection, however, the clinical picture is more distinctive, and one or other of the following clinical features is likely to predominate: swinging temperature with rigors, peritonitis, collapse, jaundice or renal inhibition.

Progressive anæmia, due apparently to deficient blood formation, is a characteristic feature of most established infections, and more particularly of those of the severer grades. In grave cases the hæmoglobin level may fall by 40% to 50% in a few days, and blood transfusions may have to be repeated on many occasions.

It must be emphasized that in four out of every five cases of infection due to anaerobic streptococci other

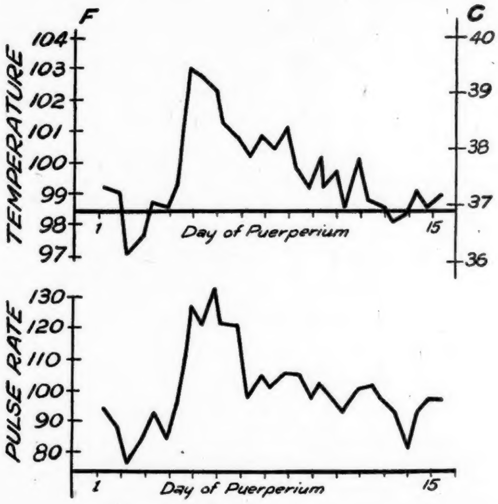

FIGURE IV.

bacteria are present in association, and their contribution to the clinical picture, although uncertain, cannot be disregarded.

Infections due to anaerobic streptococci are not infectious from case to case.

Infections due to Streptococcus Hæmolyticus Group A.

Infections due to *Streptococcus hæmolyticus* group A are the most highly infectious of all puerperal infections, and this is their outstanding characteristic. With the sole exception of infections due to *Staphylococcus pyogenes*, no other bacterial type of puerperal infection has a comparable ability or tendency to spread.

Infections due to *Streptococcus hæmolyticus* group A are apt to appear "out of a clear sky". In many such cases during labour there has been no manipulation, no instrumentation, not even a vaginal examination. The "clear sky" from which infection descended has been air contaminated with spray, droplets or dust.

The chief factor determining severity of infection is the virulence of the organism, and this can be estimated by a study of bacterial capsulation. A poor inflammatory response of the body to invasion is characteristic and is the reason for insistence on absolute rest in the treatment of these infections.

When, with highly invasive strains, general peritonitis or septicæmia develops, the onset is sudden and the acuity great, and the accompanying high fever and tachycardia tend to be of the remittent or continuous type.

The lochia are not offensive in infections due to *Streptococcus hæmolyticus* group A alone, but as 50% of these patients also harbour other bacteria, most often anaerobes, in the genital tract, the presence of offensive lochia does not remove the possibility of infection with *Streptococcus hæmolyticus* group A.

Infection due to Hæmolytic Streptococci of Groups other than A.

Infections due to hæmolytic streptococci of groups other than A are not common, but they are important because of their danger of producing infective endocarditis. They are not infectious from case to case.

Infections due to Staphylococcus Pyogenes.

Infections due to *Staphylococcus pyogenes* are not common, and in comparison with the part played by *Staphylococcus pyogenes* in wound infections in general their place in puerperal infections is a modest one.

The majority of puerperal staphylococcal infections are mild; but it is characteristic of the more severe grades that a considerable proportion are septicæmic.

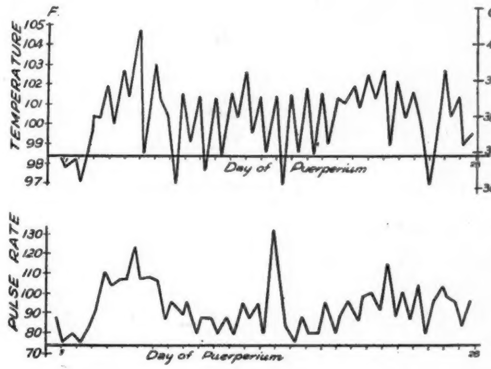

FIGURE V.

A further important feature of staphylococcal infections is their infectiousness. Although in our experience they are not so highly infectious as those due to *Streptococcus hæmolyticus* group A, others have found them extremely so, and when once established in an obstetric unit they are difficult to eradicate (Knott and Blaikley[3]). The danger of staphylococcal infections to babies in nurseries cannot be too often emphasized.

Infections due to Clostridium Welchii.

Infections due to *Clostridium welchii* are extremely rare, and most obstetricians will probably not encounter a single serious case in a lifetime. The overwhelming proportion of *Clostridium welchii* infections in women follow criminal abortion.

Serious infections occur in three chief forms: with rapidly progressive hæmolytic jaundice, with collapse proceeding to profound prostration, or with true uterine gangrene. In all these types the mortality rate is high. Infections due to *Clostridium welchii* are not infectious from case to case.

Correlation between Clinical Picture and Bacterial Cause.

The descriptions just given of the clinical aspects of the more important bacterial types of puerperal infection indicate that in general the clinical picture of puerperal infection does not show sufficient variety to suggest the nature of the infecting agent. The majority of patients merely present varying degrees of fever and tachycardia, and less malaise than the signs would lead one to expect. It is only in some of the graver infections that the clinical features are sufficiently colourful or dramatic as to indicate the probable infecting organism. In the average case a

knowledge of the factors which led to infection is a better guide to bacterial type than is the clinical picture.

DIAGNOSIS.

Puerperal infection is by far the commonest cause of fever in the puerperium, and fever is its most constant sign. The other chief causes of puerperal fever are mastitis, pyelitis and intercurrent infection, but these are not in our experience responsible for more than 25% of cases. (See Table V.)

TABLE V.

Causes of Pyrexia in the Puerperium in 1,553 Cases at the Women's Hospital, Melbourne.

Cause of Pyrexia.	Number of Cases.	Percentage.
Puerperal infection	1,184	76·2
Mastitis	132	8·5
Pyelitis	119	7·7
Intercurrent infection	118	7·6

On rare occasions puerperal infection appears without fever, but with such signs as jaundice, tachycardia, collapse or vaginal discharge.

It must be remembered that puerperal infection and other causes of puerperal fever may coexist, and the presence of an extragenital cause of fever does not remove the advisability of investigating the patient for puerperal infection.

Investigation of a Case of Suspected Puerperal Infection.

Any of the following signs in the puerperium may be due to puerperal infection and calls for an immediate clinical and bacteriological investigation: (i) a tempera-

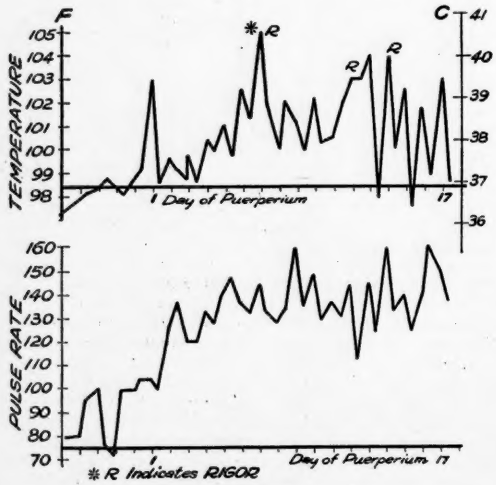

FIGURE VI.

ture of 101° F. or higher; (ii) the persistence of a temperature of 100° F. or more for twenty-four hours; (iii) jaundice; (iv) unexplained tachycardia; (v) an offensive or purulent discharge.

The routine investigation of such a case is carried out as follows.

In addition to a careful consideration of the patient's present symptoms, care is taken to elicit any past record of febrile disorders of a persistent or recurrent type, such as chronic pyelitis or tuberculosis. The important obstetric factors refer to the present labour, the conditions under which it was conducted, its duration, the time at which the membranes were ruptured before delivery, the nature and extent of operative or manipulative interference, and the fatigue, tissue damage and blood loss suffered by the patient.

Clinical examination of the patient should be thorough and gentle. Particular attention is paid to the nose and throat, lungs, breasts, kidneys, abdomen, limbs and skin. In this way the presence of respiratory infections, alimentary or systemic disorders, general infectious diseases, mastitis or pyelitis can usually be quickly determined. No vaginal examination is made in the first week.

The bacteriological investigations are in many ways the most important part of the field of inquiry. Their scope varies with the needs of the individual case and the facilities of the obstetric unit. The minimal requirements in every case are the examination of smears from vaginal swabs and attempts at aerobic and anaerobic culture of bacteria from these swabs, and the microscopic and cultural examination of a catheter specimen of urine.

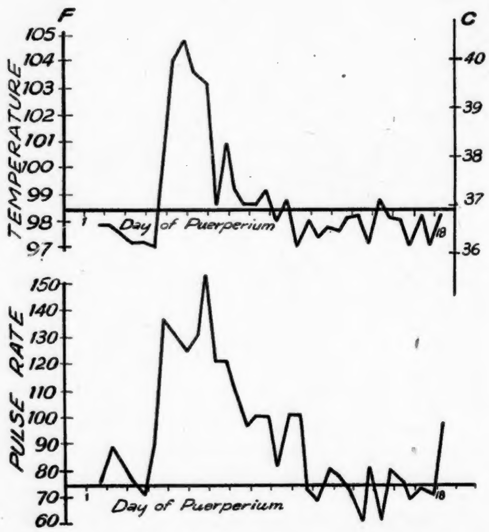

FIGURE VII.

Vaginal swabs are taken from the posterior wall with the *labia minora* held well apart during insertion and withdrawal of the swab.

In our experience an accurate bacteriological diagnosis can be made in at least 90% of cases by the examination of suitably stained vaginal smears. The great value of this particular method of investigation is that the result can be obtained immediately. Smear examination reveals not only whether infection of the genital tract has occurred, but also which types of bacteria are causing the infection. The cultures made from the vaginal swab provide confirmation of the smear findings.

In certain cases additional bacteriological investigations should be undertaken. If the patient has a sustained temperature of 102° F. or higher, or is gravely ill, and especially if the vaginal smear suggests a mixed infection, attempts at culture of bacteria from the blood may prove of value as a guide to treatment. If the patient has a sore throat or respiratory infection, or a genital tract infection due to *Streptococcus hæmolyticus* group A, throat swabs should be taken for cultural examination. If infection complicates operative obstetrics, attempts at culture should be made from any material obtained from the infected site. Intracervical smears should be prepared if *Clostridium welchii* is a possible cause.

Prevention.

The prevention of puerperal infection requires a full knowledge of its predisposing and initiating causes, and a rigid attention to detail in the measures to combat them.

Preventive measures are of two distinct types—those employed by the community, and those applied in the individual case by the obstetrician and others in attendance.

Communal Measures.

Among communal measures, the first requirements are efficient ante-natal clinics and almoner services, which should be available to all patients.

In Australia today midwifery is seldom conducted in the patient's home, for it is generally realized that it is not possible to obtain conditions suitable for the performance of completely efficient midwifery outside hospital units.

The hospital midwifery unit should be housed in a separate building, and the resident medical and nursing staffs should be trained in midwifery and their duties should be confined to that unit only. "Suspect" wards should be available for the reception of patients suspected of infection before, during or after labour, and isolation facilities should be provided for the transfer of patients proved to be infectious.

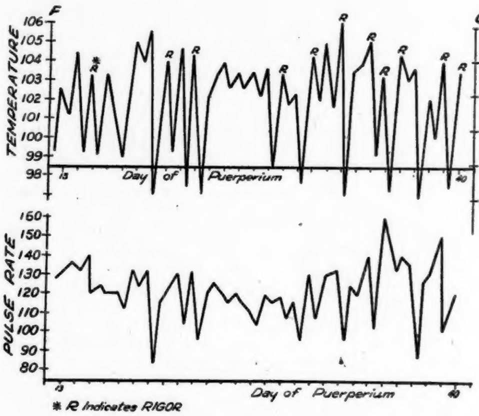

Figure VIII.

We believe that "suspect" wards can safely be contained in the main midwifery unit, but that isolation facilities must be in a separate block some distance away and conducted by an entirely separate staff.

All laundry, including blankets and other woollens, should be adequately sterilized. In the delivery rooms and nurseries, even if nowhere else, some form of air purification is desirable and will no doubt be part of a routine midwifery service in the future.

At the present time much could be done to minimize the danger of air-borne infection by the oiling of blankets and floors and by the elimination of dry dusting and sweeping.

Care of the Individual Patient.

In the ante-natal period the aim is to bring the mother to the best possible state of physical and mental health in preparation for labour and the puerperium.

Of first importance are the correction and treatment of maternal malnutrition and anæmia, of mental and bodily fatigue in the last weeks, of vaginal discharges, of respiratory and intercurrent infections, and of skin lesions such as impetigo, paronychia and furunculosis. In patients with cardiac lesions the elimination of dental infection is important. The mother should be made aware of the dangers of infective processes in herself and her family, and should be warned to avoid them and to report their occurrence.

Conduct of Labour.

During labour the aim is careful obstetrics, with a minimum of manipulative and operative interference.

In this country the greater proportion of midwifery is performed by attendants who have not received a post-graduate training in obstetrics. As a result there is considerable variation in the methods used and the standards attained, and this is particularly evident in two every-day obstetric procedures—the use of masks and aseptic technique.

In the use of masks, common errors are: (i) the use of a material which in quality or in number of layers is unsuitable to arrest all droplets that impinge on it from the respiratory tract; (ii) failure of the mask to cover the nose as well as the mouth, or to pass well under the chin; (iii) the injudicious handling of the mask by the wearer, or its removal within close proximity to the mother to enable the *accoucheur* to aspirate mucus from the baby's naso-pharynx with a catheter.

Efficient face masks should be worn by all present at the confinement, by the mother herself if she has a respiratory infection during labour, and by the attendants at every exposure of the vulva for the first week of the puerperium. Masking must never be used as an excuse to keep on duty a person whose throat is known to be infected.

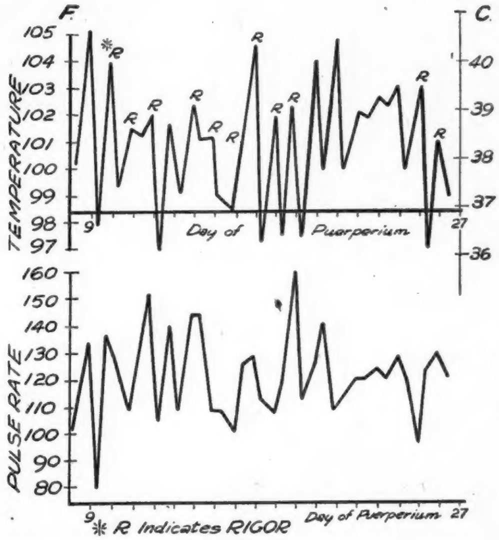

Figure IX.

Although it is generally accepted that the aseptic technique of midwifery should be of a similar order to the aseptic technique of elective surgery, this ideal is seldom realized. The following are common errors: (i) perfunctory preparation of the obstetric field; (ii) the wearing of short-sleeved gowns; (iii) the use of guards, sheets or towels which are inadequate in area, deficient in quality and applied in single thickness only; (iv) failure to discard as unsterile coverings which have become soaked in blood or liquor; (v) failure to prevent contamination from the rectum or to achieve exclusion of the anus from the field during the repair of genital wounds; (vi) failure of the obstetrician himself to complete the patient's toilet and to cover the field at the conclusion of the third stage of labour; (vii) reliance on antiseptics which are inefficient either in quality or in strength and manner employed. Of the antiseptics at present available, we have found the most generally efficient to be "Zephiran Concentrate", used in a strength of not less than one part in 160, and in the complete absence of soap. Others of value are "Dettol", used at one-third full strength, and a 1% solution of Bonney's blue.

Prophylactic Chemotherapy.

It is unfortunate that, except in the few cases in which a vaginal discharge suggests the possibility, there are no clinical aids to the detection of potential genital infection.

However, it is our experience that approximately 50% of women come into labour with potential or actual pathogens in the genital tract. As the interval after the rupture of the membranes lengthens, and particularly when labour is prolonged, the danger of intrauterine infection of the mother and baby steadily rises. Some bacteria, such as the anaerobic streptococci, show their chief effects in the mother; others, such as fæcal bacteria, are more likely to produce serious effects in the infant.

It is therefore necessary, if the mother and baby are to be afforded the greatest possible safety during and after labour, that the vaginal flora be investigated in the case of long-standing rupture of the membranes or of prolonged labour, or in the presence of a vaginal discharge, and whenever surgical measures are contemplated. Appropriate chemotherapy as indicated by the bacteriological findings can then be instituted and fully applied for the five to seven days which are necessary even in the absence of clinical infection. In the presence of lacerations or incisions of the genital tract, local chemotherapy is advisable in addition to systemic chemotherapy. Both penicillin and sulphonamides are generally applied as a powder for local application, except in contact with the peritoneal surfaces, where, in the case of sulphonamides, only solutions are used. In the presence of the clinical conditions described, and in the absence of adequate facilities for bacteriological investigation, the greatest safety for the mother and baby will result from prophylactic chemotherapy with penicillin and a sulphonamide combined. If only one form of chemotherapy is selected, penicillin should be the choice. We condemn the practice, still followed by some practitioners, of prescribing sulphonamides almost as a routine measure at the end of labour; even in established infection there are relatively few indications for sulphonamides, and their injudicious use may be dangerous.

Prevention of Spread.

At the first suspicion of puerperal infection it would be ideal practice to isolate the patient and those members of the staff who have attended her. However, complete isolation of every patient would create innumerable staff difficulties, and in Victoria at least would be unjustified on scientific grounds. As has already been pointed out, infectious cases are here very much in the minority—in our experience less than 10% of the total.

Where adequate facilities are available for immediate bacteriological diagnosis, isolation can be safely reserved for the patient proved to be infectious. Where the bacteriological facilities are not such as will enable the bacterial nature of the patient's infection to be determined within a few hours, it is advisable that those attending the infected patient should not attend other patients, and also that no patient should be admitted to the unit involved until the non-infectious nature of the infection has been established or until the infected patient has left hospital and adequate disinfection of the staff and effects has been carried out.

If the infectious nature of the case has been established, complete isolation of the patient should be enforced and an attempt made to ascertain and remove the source of the infection. Efficient disinfection of the staff, quarters and equipment should also be carried out on the patient's discharge from hospital.

Post-Natal Care.

In the post-natal period the same standards of asepsis and antisepsis employed during labour should be applied at each exposure of the genitalia and during the dressing of wounds. For maximal safety, during the first week masks should be worn by the nursing staff and by all who visit the patient.

TREATMENT.

The first essential in treatment is good nursing. The patient's room should be well aired and preferably open to sunlight. The diet should be liberal and nutritious, and the addition of vitamins *A* and B_1 is probably of value. One should aim at a fluid intake of at least five pints per day, and patients with grave infection, repeated vomiting or dehydration may require the intravenous administration of glucose.

The patient is nursed in Fowler's position to aid uterine drainage. The bowels may be assisted with bland aperients, such as "Agarol" or "Petrolagar"; but purging must be avoided. Sufficient rest and sleep are of the utmost importance and if these are not procurable with such drugs as chloral or the barbiturates, opiates should be used.

Anæmia is very common and is the clinical sign which is most often overlooked or underestimated. Any infected patient whose hæmoglobin value is 70% (Sahli) or lower is best treated by blood transfusion, which should be repeated if necessary to bring the hæmoglobin level to between 90% and 100%. Massive doses of ferrous iron together with hydrochloric acid are also advisable as a routine measure.

Chemotherapy.

Modern chemotherapy has greatly simplified the treatment of puerperal infection. But success in therapy still requires an accurate bacteriological diagnosis and stringent observance of the following principles. Whether the chemotherapeutic agent is penicillin or a sulphonamide, the initial dose should be large, an adequate concentration should be maintained throughout, and therapy should be continued for two or three days after apparent clinical cure.

The bacterial indications for chemotherapy and the methods of administration are as follows. Penicillin should be used in the treatment of infections with *Staphylococcus pyogenes*, anaerobic streptococci, sulphonamide-resistant group A hæmolytic streptococci, *Streptococcus hæmolyticus* group B and *Clostridium welchii*. Penicillin has five methods of administration—intravenous, intramuscular, subcutaneous, oral and local. The intravenous route is rarely used in Australia, owing to the difficulty of obtaining sufficiently pure penicillin. There are three methods of administering penicillin intramuscularly. (i) Intermittent injection is the usual mode. Doses varying from 15,000 to 40,000 Oxford units may be given every three hours, and the dosage and intervals may increase up to as much as 100,000 units every twelve hours. (ii) The continuous drip method consists in the administration of 100,000 to 150,000 Oxford units per day in 4·0 to 20 ounces of normal saline solution. (iii) Penicillin may be given in suspension—150,000 to 300,000 Oxford units every eighteen to twenty-four hours in a mixture of beeswax (6%) and peanut oil. If the subcutaneous route is used, 50,000 to 100,000 Oxford units of penicillin are given every eight to twelve hours. The oral route is the least reliable and is five times as extravagant as the others. Locally, penicillin may be applied as a powder, in the proportion of 50,000 to 100,000 Oxford units with 10 grammes of sulphanilamide, for the treatment of incisions and lacerations.

Sulphonamide therapy is required in infections with *Streptococcus hæmolyticus* group A, *Bacterium coli* and anaerobic Gram-negative bacilli. The chief drugs are sulphanilamide, sulphadiazine, sulphacetamide (all given every four hours) and sulphamerazine, given every twelve hours. The principles of dosage are as follows. A large initial dose should be given (two to four grammes), an adequate blood concentration should be maintained (eight to fourteen milligrammes per 100 millilitres), and administration should [illegible] for two or three days after clinical signs have subsided. Alkalis and copious fluids must be given [illegible] urinary output, the red and white cell counts and [illegible] should be carefully watched. As a guide to dosage [illegible] suggested that in order to maintain a concentration [illegible] blood of 10 milligrammes per 100 millilitres, one gramme [illegible] every 20 pounds of body weight should be given every twenty-four hours.

We still believe that in grave infectious requiring penicillin the best results are obtained by its administration

every three hours. In grave infections requiring sulphonamide therapy our present practice is to use sulphamerazine or sulphadiazine, together with alkalis. Where a sulphonamide is indicated in the presence of renal damage we employ either a combination of two or more sulphonamides or sulphacetamide because of its very great solubility.

Treatment of Individual Lesions.

Modern chemotherapy has reduced the need for treating individual lesions.

For pelvic infections heat is of value. In the early stages it is applied externally for the relief of pain, and later in the form of medical diathermy or ultra-short-wave therapy to hasten resolution in resistant cases.

The formation of a localized peritonitic or cellulitic abscess, or the establishment of general peritonitis, calls for surgical drainage. Local as well as systemic chemotherapy can then be continued in the form determined by bacteriological examination of the released fluid. In general peritonitis especially, other aids include opiates, continuous intravenous therapy and on occasion the indwelling duodenal or Miller-Abbott tube.

Nowadays the ligation of thrombosed pelvic veins is rarely practised, and the indications for hysterectomy have been almost eliminated by the availability of scientifically directed chemotherapy.

The Problem of Puerperal Infection Today.

Although puerperal infection is not so grave or so common a problem today as it was fifteen years ago, its incidence and death rate must be regarded as still too high, particularly when it is remembered that in recent years conditions have been most favourable for attacking this problem. Knowledge of the causes of puerperal infection is exact, detection of the infecting bacteria in individual cases can be speedy and accurate, and the obstetrician has at his service, in the great majority of cases, potent chemotherapeutic agents.

In our opinion the chief factors retarding progress are insufficient appreciation on the part of medical and nursing personnel of the specific causes of puerperal infection, inadequate attention to detail in practice, and the absence of proper facilities for the immediate bacteriological detection of infection.

We would emphasize the following points. It is still not sufficiently realized that puerperal infection is divisible into two distinct groups, the infectious and the non-infectious, and that the former group is small by comparison. Any measures directed towards prevention and control which do not appreciate this significant division are obsolete and unreal.

In the everyday treatment of puerperal infection, the two commonest errors are lack of early and adequate bacteriological investigation and the misuse of chemotherapy. The elimination of these errors depends on the provision of a first-class bacteriological service and full cooperation between clinician and bacteriologist. At the present time an integrated service of this nature is available only to occasional patients and to isolated obstetric units; but it should be available to the community at large.

With the proper application of modern knowledge and modern methods the control and management of puerperal infection should pass more and more into the realm of preventive medicine.

References.

(1) H. M. Butler and A. M. Hill: "Hæmolytic Streptococcal Infections following Childbirth and Abortion. I: Determination of Virulence of Group A Strains", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1940, page 222.

(2) H. M. Butler: "Bacteriological Studies of Clostridium Welchii Infections in Man", *Surgery, Gynecology and Obstetrics*, Volume LXXXI, 1945, page 475.

(3) F. A. Knott and J. B. Blaikley: "The Control of Staphylococcus Aureus Infections in a Maternity Department", *The Journal of Obstetrics and Gynæcology of the British Empire*, Volume LI, 1944, page 386.

Reviews.

THE INSPECTION OF FOODS.

FOUR years ago we reviewed favourably the first edition of "Food Inspection Notes"[1] which we considered would be found quite useful as an epitome of the subject by medical men who were not full-time officers of health and yet might be called upon at short notice to inspect foodstuffs. In size, the book will readily go into the pocket, and its contents can be quickly consulted. It is also the kind of book that medical officers of health can recommend to their health inspectors, who, especially in country districts, may have many other duties to perform besides an oversight on foods and are likely to welcome such a handy epitome of the subject at a very reasonable price.

The book has been prepared by two practising public health officials, and the fact that a second edition has been called for so soon shows that it was wanted and has been successful. It has, of course, been compiled for English inspectors more particularly and adjustments for Australian conditions or requirements may have to be made. The book begins with meat inspection where the slaughtering and dressing of carcasses and their inspection are accompanied by such useful anatomical details as the location of the lymphatic glands in the ox, sheep and pig; then comes a description of the diseased conditions that may be found and the appearances of imported meat, and the methods of inspection of meat by-products and of poultry, game, fish and shell-fish. Then come accounts of milk and milk products, canned and other foods, and an epitome of food-poisoning and the methods of taking samples. The work still requires revision by a medical or veterinary officer to eliminate such slips or errors as that on page 31, where malignant tumours are described as "encapsuled", and the statement is made that swine erysipelas is not communicable to man (a fatal case with vegetations on a heart valve has occurred in Australia, but this, of course, is very rare). The female *Onchocerca* worm in Australian "worm-nests" in cattle is longer than one inch. It reads as though hydatid cysts with daughter cysts were mistaken for *Echinococcus multilocularis*. In the life-history of flukes *Reclia* should be *Redia*. In spite of these little blemishes, the book is undoubtedly a useful one for its particular purposes.

RHEUMATISM AND SOFT TISSUE INJURIES.

IN his book "Rheumatism and Soft Tissue Injuries" James Cyriax has supplied a well-felt need in the medical world which should be of very great value to the general practitioner and young graduates, whilst it may initiate a reappraisal of diagnostic and therapeutic methods as practised by specialists in physical medicine and rheumatology.[2]

The time-honoured work of Hilton on the necessity for rest in the treatment of pain comes under scrutiny, and the author postulates the advisability of movement in the therapy of pain, a new concept referable to soft tissues. The dermatomes are given in detail, their knowledge being so essential for the correct elucidation of referred pain.

After dealing with the differentiation between neuritis and perineuritis, the author unfolds in the fourth chapter one of the foundations upon which the book has been built, namely, the diagnosis of soft tissue lesions. In this regard he makes special mention of the history, local examination, active, passive and resisted movements and the importance of local anæsthesia. With this mosaic of diagnostic essentials localization of soft tissue lesions passes out of the realm of inexactitude into that of specific localization, with sequential orderly direction of appropriate treatment.

Local anæsthesia is utilized as a confirmative measure to diagnostic accuracy, as well as for its therapeutic efficiency, and the sphere of usefulness for procaine is thus widened.

The definite attempt to pin point the soft tissue lesion is the aim of the author, and the methods he has employed so sedulously and with success are a contribution to the clinical side of physical medicine. Diagnostic accuracy is the keystone to successful therapy in soft tissue lesions,

[1] "Food Inspection Notes: A Handbook for Students", by H. Hill, F.R.San.I., F.S.I.A., A.M.I.S.E., and E. Dodsworth, M.R.San.I., M.S.I.A.; Second Edition; 1947. London: H. K. Lewis and Company, Limited. 6½" × 4", pp. 132. Price: 6s.

[2] "Rheumatism and Soft Tissue Injuries", by James Cyriax, M.D., B.Ch. (Cantab); 1947. London: Hamish Hamilton Medical Books. 9½" × 6", pp. 410, with many illustrations. Price: 42s.

but the arch of successful therapeutics is built up to a great extent by friction (deep) massage and local anæsthesia.

The following twelve chapters are occupied by the description of lesions in various parts of the body. It is in these that we observe the clinician at work, localizing the different lesions in specific structures, differentiating one from the other by the basic principles of diagnosis laid down earlier. Here we see a wealth of clinical knowledge gleaned from the ripened fields of wide and long experience; here, the author speaks with authority, showing by numerous diagrams and photographs the localization of specific lesions and the administration of friction (deep) massage, ensuring recovery of movement, a message he has so ably propounded. Though many may doubt the degree of therapeutic efficacy claimed, there must be few who are capable of delivering judgement.

In the chapter dealing with the diagnosis of backache many provocative opinions are expressed, divergent from current beliefs, but bearing the hall-mark of careful observation and being the result of painstaking localization. The introduction of a term "dural pain" in the description of pressure by a fragment of the intervertebral disk on the central part of the contiguous dura is new in medical literature, and the author further postulates that this is a common cause of lumbago, being relieved by epidural local anæsthesia.

In the two chapters on treatment by movement he reveals the difference between ordinary massage and deep massage, the latter denoting deep transverse friction, that is, across the fibres of muscle or tendon. This is applied to a specific area with technical exactitude "the right spot in the most effective way". He stresses the value of passive, active and resisted movement, indicating their precise usages.

Local anæsthesia in the treatment of injuries is ably dealt with, the knowledge of its effectiveness now being widespread. Short-wave diathermy is described as the best form of heat therapy, whilst some scorn is cast on the popular infra-red ray therapy. The morbidity of the rheumatic diseases in Great Britain is shown to be very high, whilst the paucity of facilities and trained staff, a state of affairs duplicated in Australia, is deplored.

The lack of adequate teaching of medical students, the relative ignorance of many medical practitioners, and the need for the profession to gain knowledge of the diseases considered, especially as regards their diagnosis and treatment, is indicated in the final chapter.

The book is easy to read, being well illustrated by numerous diagrams and excellent photographs.

THE ISOLATION AND IDENTIFICATION OF POISONS.

THE first edition of Bamford's book on poisons was published in 1940 and was a very welcome addition to the literature on this subject.[1] For many years there had been a need for a concise work on poisons, giving their characteristics, methods of isolation and purification, and a systematic scheme for their identification. This need has been met by Bamford's book. The second edition revised by C. P. Stewart, M.Sc., Ph.D., follows the same method of presentation as the first edition. The additions which have been made are most useful from a practical point of view.

The information given and the methods described in the first edition have proved to be reliable and the second edition can be recommended to practical workers in this field.

INTRACRANIAL COMPLICATIONS OF EAR, NOSE AND THROAT INFECTIONS.

IN the preface to his book, "Intracranial Complications of Ear, Nose and Throat Infections", Hans Brunner assumes that the reader will be "acquainted with the principles of oto-rhino-laryngology and neurology".[2]

This volume comprises more than 400 pages, and, despite fairly numerous typographical errors and at times a somewhat stilted usage of the English tongue, it is well produced and contains many good (but not coloured) photographs, illustrations of sections, diagnostic studies and drawings of various pathological conditions.

In an excellent introductory section written in narrative form, the anatomy and, to a lesser extent, the physiology of the *dura mater*, dural sinuses, pia-arachnoid, intermeningeal spaces, cerebro-spinal fluid and brain, are discussed. This should prove equally interesting to both ear, nose and throat surgeons and neurosurgeons. The remainder of the book is devoted to the various intracranial complications, including most excellent descriptions of petrositis and brain abscesses.

In the various intracranial complications the pathology, symptomology, prognosis and treatment are discussed.

Most standard operative measures are mentioned, but, in the field of chemotherapy, the author freely states that, so far, the value of penicillin and the newer antibiotics has not been finally assessed.

This book will undoubtedly be accepted as giving the most modern and up-to-date concept of intracranial complications due to the diseases of the ear, nose and throat, and future editions will be able, in the course of the next few years, to give a more complete evaluation of chemotherapy in these cases. The work should be a valuable addition to the library of all ear, nose and throat surgeons, and be used as a textbook by those studying this specialty.

[1] "Poisons: Their Isolation and Identification", by Frank Bamford, B.Sc.; Second Edition revised by C. P. Stewart, M.Sc., Ph.D., with a foreword by Professor Sydney Smith, C.B.E., M.D., F.R.C.P.; 1947. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 312, with illustrations. Price: 21s.

[2] "Intracranial Complications of Ear, Nose and Throat Infections", by Hans Brunner, M.D.; 1946. Chicago: The Year Book Publishers, Inc. 9¼" × 6¼", pp. 456, with many illustrations. Price: $6.75.

Notes on Books, Current Journals and New Appliances.

THE BRITISH JOURNAL OF NUTRITION.

THE first number of *The British Journal of Nutrition* has appeared. It has been published by the Cambridge University Press for the Nutrition Society. It will be devoted to reports of original work carried out in all branches of nutrition and a volume will consist of four parts appearing at quarterly intervals. *The Proceedings of the Nutrition Society* will be merged with the new journal. The Nutrition Society, it should be noted, has as its object the advancement of the scientific study of nutrition and its application to the maintenance of human and animal health.

The contents of the first issue are interesting and cover a wide field. To begin with there are obituary notices of Sir Joseph Barcroft and Sir Frederick Gowland Hopkins. Then follows a paper by L. J. Harris and L. W. Mapson on the determination of ascorbic acid in the presence of interfering substances by the "continuous flow" method. S. J. Folley and others write on reproduction and lactation in the rat on highly purified diets. T. Moore and Y. L. Wang discuss the formation of fluorescent pigment in vitamin *E* deficiency. R. Brande and others report a study of the composition of sow's milk. M. Pyke describes the nutrient content of English prison diets in 1944. There is a report of a conference on education in nutrition and the journal closes with abstracts of communications to two meetings of the Nutrition Society.

THE ADMINISTRATION OF BLOOD AND SERUM.

THE New South Wales Red Cross Blood Transfusion Service has built up a sound organization for the provision of stored blood and serum and for the testing of blood donors. In the process much useful information has been acquired. A booklet[1] has now been issued by the service which sets out details regarding the service provided including notes on the care and selection of donors; brief but comprehensive discussions on the place of intravenous therapy in trauma, post-operative shock, burns, hæmorrhge and anæmia; and the essentials regarding reactions to transfusion, blood typing (including the Rh factor) and the technique of intravenous infusion. The material is up to date and worth careful perusal. The booklet is available free from the headquarters of the service at 374, George Street, Sydney.

[1] "Administration of Blood and Serum: Principles, Apparatus and Techniques"; 1947. Sydney: Red Cross Blood Transfusion Service, New South Wales. 8½" × 5½", pp. 44, with illustrations. Distribution gratis.

The Medical Journal of Australia

SATURDAY, FEBRUARY 21, 1948.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

HOCKING VERSUS BELL.

The decision by the Privy Council in what has become known throughout Australia as the "Hocking and Bell" case has concluded one of the most protracted actions in the legal history of New South Wales. The Privy Council's judgement is published in full on page 242. From the issue of the writ to December 18, 1947, when the Privy Council delivered its judgement, nearly seven years elapsed. During this time the matter came before four separate juries, these trials taking in all sixty-one days. It was considered by the Full Court of the Supreme Court of New South Wales on three occasions, by the High Court and then by the Privy Council. As Viscount Simon remarks, it has "a regrettably long history".

The mass of evidence is very shortly summarized in the Privy Council's judgement, but it was commented on in more detail by the five Justices of the High Court as reported in this journal on February 23, 1946. Accordingly, any further statement here of what was put to the several juries by the many witnesses and the arguments submitted to the appellate courts is uncalled for. The case has had much publicity in the daily Press and it has excited great interest, particularly among the members of the medical profession. The question uppermost in the minds of practitioners is the effect of Hocking *versus* Bell on future actions that may be brought by patients alleging negligence against their doctors. In this case the complexity lay not in ascertaining the legal principles involved, but in applying them to a given set of facts. Viscount Simon expresses the position in these words: "If, at the end of the hearing of witnesses, the evidence is all one way, so that no jury can reasonably find for the plaintiff, and a verdict and judgment in favour of the plaintiff are nevertheless given, it is within the competence of the Supreme Court to direct that verdict and judgment should be entered for the defendant. The main question in this appeal really is whether that is the situation with which the Supreme Court had to deal. . . . He (the defendant) claims that though the plaintiff made a *prima facie* case in the first instance, the evidence subsequently called established beyond dispute that what the plaintiff alleged and swore to have happened could not possibly have happened, and, this not being an age of miracles, the defendant must succeed. Their Lordships must therefore proceed to examine the testimony for the purpose of seeing whether, on the evidence taken as a whole, this is the resultant position."

The evidence of Mrs. Hocking was, to say the least of it, most extraordinary. The Privy Council describes it as "remarkable", and, in some of its detail, "strange and surprising". In the High Court, Mr. Justice Dixon, though he dissented from the majority, referred to "a natural incredulity concerning the plaintiff's story". A judge in the Supreme Court spoke of its "improbability". It should also be noted that some of her evidence was of such a nature that it could neither be corroborated nor contradicted. In short, so long as New South Wales law remains unaltered, if a patient sues a doctor for negligence and gives evidence in the witness box which, though improbable, is sufficient to raise a *prima facie* case and some expert opinion is called to support the possibility of her case, if then the plaintiff obtains a verdict from the jury, it would be imprudent to pursue the matter further by invoking the aid of the appellate courts however imposing the body of medical authority called to support the defendant. In these circumstances, subject to new trials being ordered, the jury reigns supreme in deciding the facts.

It is interesting to consider two possible practical results following Hocking *versus* Bell. First, in view of the tenacity with which Dr. Bell defended his professional reputation, future litigants may be deterred from facing the long and thorny path trodden by Mrs. Hocking, although it ultimately led her to £800. On the other hand, if an indemnity policy is involved, any underwriter considering a similar legal pattern would be well advised to settle rather than to fight.

If the law in New South Wales had been changed to conform to the position in England and other parts of the Empire, this case might well have produced an opposite result. It would certainly have the most important bearing on future actions of a like nature. Generally, where the judicature system applies, the Full Court before which a verdict is impugned is authorized "to draw inferences of fact and make such orders as the case may require, so that, as has been held in England, when the Court has all the facts before it, after setting aside a verdict as being against the weight of evidence, the Court may enter judgment for the other party if they think that a new trial could bring to light no further material facts, and to do so will do complete justice between the parties". The Privy Council was most careful not to express an opinion on this matter. It seems, though, after reading the observations of the New South Wales judges who considered the case, that if the Full Court had possessed the right to draw inferences of fact, it would have found for the defendant.

The jury system has played an important part in the legal system in New South Wales and it will continue to do so. Justice demands, however, that as some problems are too complex for the ordinary man, they should be decided by minds especially acute and disciplined. A passage from the judgement of Mr. Justice Dixon is significant (71 C.L.R., page 491): "There is, I think, much danger of our misconceiving and misapplying the

anatomical, surgical and medical treatises and plates, but I am bound to say that, so far as I have been able to master the relevant information they contain, it seems to me to show that many difficulties must be encountered by any hypothesis which would explain what the plaintiff says happened with the piece of tube described. I cannot, however, imagine these treatises proving of any assistance to the jury."

Whatever the merits of Hocking *versus* Bell, it is to be hoped that the law in New South Wales will be altered so that in future the power of a jury is restricted to the realm of its competence.

Current Comment.

VIRUS INFECTION.

At the Sydney meeting of the Royal Australasian College of Physicians in October, 1947, Professor F. M. Burnet, of the Walter and Eliza Hall Institute of Medical Research at Melbourne, gave an account of important work that had been carried out by him and members of his staff on cellular infection by influenza and related viruses. This work has now been published;[1] it is of the first importance and has far-reaching possibilities. Burnet summarizes his findings at the beginning of his article in the following words: "We have found that the influenza viruses contain as an integral part of their surface structure an enzyme which is adsorbed by and eventually destroys certain specific molecular groupings of a mucin which forms part of the cell surface. Unless this process can occur, infection of the cell cannot take place. We have obtained and partially purified a soluble enzyme which, by destroying the specific adsorptive power of the mucin in the cell surface, prevents adsorption of the virus and infection."

In telling the story of his work, Burnet starts with reference to virus hæmagglutination and the work of Hirst. Hirst found in 1941 that fluids produced in the allantoic sac of chick embryos by the multiplication of influenza viruses agglutinated red cells. The agglutination was found to be a function of the virus particles themselves. In 1942 Hirst showed that red cells to which virus was adsorbed in the cold could be freed of virus by being held for a few hours at a temperature of 37° C. With the elution of the virus the cells became stable in suspension and were not agglutinated by fresh virus. On the other hand the eluted virus retained its full infective and agglutinating power and could be used again to treat repeated quantities of red cells. In other words, the virus produced an effect on the cells, but was itself unchanged and ready for further onslaughts on the "receptors" of other cells. This was seen to be characteristic of enzyme action. In 1943 Hirst demonstrated similar reactions in the excised lung of the ferret. From the findings he concluded that the susceptible cells of the respiratory tract reacted in the same way as red cells and that the essential first stage in the process of infection was adsorption to and enzymic destruction of some specific component of the cell surface. Burnet then describes how he set out to identify this component of the cell surface.

In 1946 Burnet and his fellow workers formulated the idea of a receptor gradient. The viruses of the group which agglutinate red cells by the direct action of virus particles can be arranged in a linear series in such a way that red cells rendered inagglutinable by any given virus in the series are also inagglutinable by any virus which precedes it, but may be agglutinated by some or all of the viruses which follow it. Burnet refers to this work and shows how it led to the discovery of a new type of agglutinability in treated cells. He found that cells which had been acted on and stabilized by an influenza virus had become agglutinable by any serum, normal or immune, to a moderate titre such as 1:160. For an immune serum to be successful in stabilizing partially treated cells, it had to contain sufficient antibody for it to be effective at dilutions beyond 1:160. Describing his investigations from this point, Burnet refers to the phenomenon described by Thomsen in 1926—occasionally casual bacterial contaminants will multiply in a suspension of human red cells and change their character, so that they are agglutinated not only by the iso-agglutinin serum appropriate to their A B O group, but also by any other serum. Two years later Thomsen's collaborator, Friedenreich, showed that the effect was due to a bacterial enzyme which was produced by two types of bacteria —diphtheroids and vibrios. Burnet points out that Friedenreich's description of the way in which these bacterial enzymes acted indicated a close similarity to the Hirst phenomenon. He set to work to obtain a bacterial enzyme of this kind. He describes his preparation of the enzyme from filtrates from a strain of *Vibrio choleræ*. This enzyme, called the receptor-destroying enzyme and referred to always as "RDE", was antigenic, producing with appropriate immunization an antiserum which specifically inhibited its action on red cells and on other substrates. In discussing the biological activities of "RDE", Burnet refers to work by Stone (of the Walter and Eliza Hall Institute) showing that the action of "RDE" is in all essential respects similar to the action of the influenza viruses. Stone, we read, has just completed a very comprehensive study of the effect of "RDE" on virus adsorption, elution and infection in the allantoic cavity of the chick embryo. Her results are set out briefly as showing:

> (1) that in living embryos virus is adsorbed but not eluted;
> (2) that in embryos killed by washing out the allantoic cavity with formalin, adsorption and elution occur as with red cells or with the mouse lung;
> (3) that the addition of RDE prevents adsorption of virus;
> (4) that, by suitable treatment of the cavity with RDE, infection with any influenza virus or with N.D.V. [Newcastle disease virus] or of mumps can be prevented; and
> (5) that after removal of RDE there is a recovery of susceptibility (regeneration of receptors) in 24-48 hours.

Burnet points out that this investigation establishes for the first time the fact that virus infection can be prevented by the enzymic destruction of a cell constituent. It is at this point that he draws attention to the potential significance of the work in regard to the development of a method of active therapy of virus infection.

Of the utmost importance to this whole investigation is the demonstration of the nature of the substrate on which "RDE" acts. The work involved is described shortly; it was based on work by Francis on the high inhibitory power of serum against hæmagglutination by heated influenza B virus, and by work of Burnet and McCrea on the non-specific inhibitory titre of normal sera. The findings revealed the presence of a non-cellular source of substrate for the enzyme. This substrate was found to be a mucin, and evidence is brought forward to show that mucin is the primary substrate of enzyme action by influenza viruses.

Emphasis has been laid on the possible practical outcome of this work. Burnet writes: "If the particular aspect of the mucin molecule on which the virus enzyme acts can be chemically defined, real possibilities either of blocking the receptors or of inactivating the enzyme by an appropriate drug will arise." In commenting on this, *The Lancet* confesses to being less optimistic than Burnet is: "The agents which he has in mind presumably act at the cell-surface, not within the cell; and we have already, in immune sera, substances which are very potent against extracellular virus but are, to say the least, therapeutically disappointing." All that Burnet states is that the work has its "greatest potential importance" in the fact that "it opens up a possible approach to the chemotherapy of virus disease". He remarks that if certain aspects

[1] *The Lancet*, January 3, 1948.

of the mucin molecule can be chemically defined, certain real possibilities arise. This is a restrained statement. In regard to tropisms of viruses Burnet is not prepared to accept too facile an explanation. Mucin may not tell the whole story. There is no doubt that the field for future research opened up by this long and laborious work is enormous.

MAD DOCTORS.

WHEN Philippe Pinel, physician at the *Hôpital Bicêtre* of Paris soon after the French Revolution, appealed to the Common Council of Paris for assistance for the insane and for authority to use humane treatment in their management, a prominent citizen visited the hospital and questioned Pinel's own sanity because of his attitude towards his patients. Long before then and even up till today suspicion has hung heavily about those who deal with crazy people that they also are not normal. The novel writer, of course, finds the mad doctor an excellent subject with almost limitless possibilities for building up cunning stories of suspense and horror. However, the palpably mad doctor is fortunately rare and the group within the profession whom P. G. Wodehouse has so delicately termed "loony doctors" are probably more than able to cope with the gentle inuendoes and clumsy brickbats aimed at them. One psychiatrist, A. E. Bennett,[1] has taken up the old discussion and contends that "mad doctors" have not been and are not limited to the psychiatrists. He makes much play of the tendency of the profession to swing from fad to fad, carrying fads to excess and then forgetting them. He is especially critical of the attempted cure of functional nervous disorders by means of unnecessary surgery, "vitamin mania", the excessive use of sedatives, diet fads, counter measures for intestinal intoxication, gynæcological procedures and so on. Nor does he allow the psychiatrist to go free of criticism. He recalls the extremes and lack of uniformity in practice of psychoanalysis, and questions the wisdom of the currently popular label "psychosomatic medicine"—a nice sounding name, he thinks, leading to confusion. He contends that there is an unsound trend to discount the importance of neurological training for the psychiatrist, and affirms that "no competent psychiatrist can succeed without a good organic neurologic background". This last view is generally accepted in this country and should be upheld at all costs. The trend mentioned by Bennett is apparently, however, a real factor in America. Other psychiatrists of good standing in that country have discussed the matter at some length and not without apprehension, as some extremists have even suggested that a knowledge of organic disease may be harmful to a psychiatrist and may interfere with his appreciation of functional disorders!

Reverting to the subject of fads and passing fashions, Bennett suggests that so-called modern methods of treatment in psychiatry, such as convulsions, coma, lobotomy and artificial fever, may seem just as mad to our successors as do the methods of earlier psychiatrists to us now. He contends that the irresponsible methods by which some of these procedures are now being applied are certainly madness. Excessive convulsive and prolonged coma treatments for those with chronic incurable schizophrenic conditions is to be condemned, and it is very difficult to justify the use of these drastic measures as consulting room procedures, if only for the reason, as Bennett points out, that patients sufficiently ill to need such treatment should have hospital care.

Bennett's article is salutary if it is viewed in the right way. He has covered a great deal of ground and has dealt not only with simple craziness, but also with a more reprehensible kind of madness hard to distinguish from iniquity. When we think of fads and fashions we have to distinguish between commendable zeal and repetition growth into a habit. They are extremes, but a dividing line must be found by each practitioner. Even so there is madness and madness; sometimes, like the madness of Hamlet, there's method in it. What is needed is that we seek, each of us, in the mirror of common sense some reflection of our tendencies. Bennett would surely agree with this and no doubt like a wise man he seeks his own reflection from time to time. Some of us have friends who think so well of us that they will tell us the truth and try to strip our craziness from us. If we have we are fortunate.

[1] *The Journal of Nervous and Mental Disease*, July, 1947.

THE TREATMENT OF MULTIPLE MYELOMA.

MULTIPLE MYELOMA is a disease which has attracted a good deal of attention in spite of its comparative rarity. The hyperglobulinæmia which accompanies it is sometimes associated with the phenomenon described by Bence Jones and bearing his name, a curious finding well known to most medical students, even though they have never actually seen it. One of the latest suggestions for ameliorating the hapless condition of the unfortunate subjects of this disease was inspired by this association of excess of a particular protein group in the blood. Michael A. Rubinstein points out that there are several other diseases in which hyperproteinæmia and hyperglobulinæmia occur, kala-azar, *lymphogranuloma venereum* and schistosomiasis.[1] These are widely different in nature, though the ætiological agent is known in each case, whereas multiple myeloma is in many ways a mystery, lying somewhere on the vague boundary between other forms of neoplastic disease and blood dyscrasias. Rubinstein remarks that this apparently unrelated trio have two common denominators, the frequent occurrence of hyperglobinæmia, which is presumably bound up with somatic involvement, and the favourable response to the same therapeutic agent, antimony. Accordingly he has made the suggestion that the success of antimony may be due to some biochemical characteristic of the three different types of infection, and he thought it worth while to investigate the effect of antimony in myeloma.

This idea is not completely new, for Snapper in 1945 tried the action of stibamine in myeloma, on the basis of its success in kala-azar. Rubinstein's work has now been in progress since 1943, and he has during this time tried a number of antimony compounds. The toxicity of tartar emetic was found a drawback, and after other salts had been used "Neostibosan" was found more suitable. Great care is necessary, particularly if renal involvement is present, and the blood and urine are frequently examined. A full course consists of 15 grammes in divided doses of 0·3 gramme given at appropriate intervals. Seven patients have so far been treated. Naturally it is hard to evaluate the results of any treatment in a disease that is uncommon, subject to remissions, and responsive to X radiation in variable degree. Examination of the bone marrow showed that following treatment with antimony the plasma cells were frequently decreased in number, and also showed well-marked metachromatic basophilic granulation. The author admits that spontaneous changes may occur; he was struck by the apparently selective action of the drug on the plasma cells, for no other marrow elements underwent change. More important is the observation that the tumour masses and infiltrations were much more radiosensitive after the treatment. Rubinstein is cautious about claiming that this is necessarily an effect of the drug, but so far the indications are encouraging that antimony may exert some influence on the marrow cells. If such influence enhanced the sensitivity of the myeloma tissue to X rays, it would be a definite advance in the handling of a disease which can cause such crippling and painful disability. No definite changes were observed in the hyperglobulinæmia incidental to the disease that could be attributed to antimony. Perhaps the idea underlying this method of treatment is illogical, but there is discernible in therapeutic research today an effort to find some correlation between the effects of known or unknown agents of disease on the chemistry of the body and the ascertained effects of drugs of known composition.

[1] *Blood*, November, 1947.

Abstracts from Medical Literature.

RADIOLOGY.

Osteochondritis Dissecans.

GERALD LAVNER (*American Journal of Roentgenology*, January, 1947) states that *osteochondritis dissecans* is a non-infectious lesion in which there is necrosis of a segment of subchondral bone with formation of an osteo-cartilaginous sequestrum. Trauma, either direct or indirect, probably plays an important role in the formation of the disease; its exact mode of origin is undetermined. The radiological appearance is characteristic; there is a sharply defined cavity from which arises a sequestrum of bone. Depending upon the age of the lesion, the sequestrum may still be within the cavity and covered by unbroken cartilage or it may have been extruded into the joint space. This condition occurs more frequently than is currently accepted. The treatment may be either conservative or surgical. In cases of the disease without symptoms, in which the sequestrum is not detached, no treatment is indicated other than immobilization. In cases without separation of the sequestrum, but with symptoms, physiotherapy has been tried, but without improvement; in these the best treatment is probably by operation. When there are loose bodies in the joint surgical removal is the only acceptable method of treatment.

Motor Dysfunction of the Biliary Tract.

A. C. IVY (*American Journal of Roentgenology*, January, 1947) states that it has been established that the *sphincter choledochus* or choledocho-duodenal mechanism can contract in man with sufficient force to prevent the evacuation of the contracting gall-bladder and to counteract the secretory pressure of bile. Convincing evidence is available showing that pain may arise both from the rather sudden distension of the biliary passages by a force within the range of the contractile power of the gall-bladder and of the secretory pressure of bile, and also from spasm of the *sphincter choledochus* and/or of the adjacent duodenum. There is adequate evidence of a physiological nature, provided by a large group of investigators, to justify the view that a motor dysfunction of the biliary passages may produce symptoms, or that such an entity as biliary dyskinesia exists. There is no sound reason to doubt today that the gall-bladder empties itself by way of the cystic duct under the force of its contraction which is caused primarily by the action of the hormone cholecystokinin. The hormone cholecystokinin is produced by the upper intestinal mucosa when it is in contact with acid, fat or partially digested protein. It is not produced in response to a carbohydrate meal or water, as judged from the lack of effect of these substances on the evacuation of the gall-bladder. In the absence of manometric studies via a choledochostomy, the clinical diagnosis of biliary dyskinesia is difficult, if not impossible, to prove. The strongest circumstantial evidence in support of the diagnosis, in the presence of a gall-bladder which can be visualized, is the visualization of the hepatic ducts with pain after the ingestion of a fatty meal. This combination has only rarely been observed. The diagnosis of biliary dyskinesia in the patient with or without a gall-bladder is a challenging radiological and clinical problem. There is reason to hope that the isolation and use of cholecystokinin will assist in the solution of the problem.

Torulosis.

ROY R. GREENING AND LEON J. MENVILLE (*Radiology*, April, 1947) observed certain X-ray findings in cases of torulosis, which, while not pathognomonic of the disease, should strongly suggest the possibility of infection by a yeast-like organism such as *Torula histolytica*. When these findings are correlated with the clinical picture, the authors believe the diagnosis should be fairly certain. The early lung lesions offer the most favourable opportunity for a satisfactory X-ray diagnosis. These were found to be fairly well circumscribed patchy areas of homogeneous consolidation, with only a small amount of reaction about their edges. These small areas of consolidation tend to become confluent as the disease progresses, with or without cavity formation, and with little or no demonstrable drainage. The lesions resemble closely those of tuberculosis. However, they tend to occur more frequently in the bases of the lung, where tuberculosis is only occasionally found. As healing occurs, small amounts of fibrosis remain in the affected areas. The lymphatic structures in the hilar region may show slight enlargement, not nearly so prominent as is seen in Boeck's sarcoid and in certain forms of pneumonokoniosis. Then, too, the associated involvement of the nervous system present in nearly all cases of pulmonary torulosis is a strong differential point against tuberculosis. The lung lesions as observed radiologically suggest a fungous infection, and, when associated with symptoms of the cerebro-spinal system, should lead to the inclusion of torulosis as one of the first-choice possibilities in the diagnosis.

Pulmonary Sarcoidosis.

L. HENRY GARLAND (*Radiology*, April, 1947) states that the pulmonary changes in sarcoidosis are one of the most conspicuous manifestations of this disease. The thoracic radiological findings may be divided into those showing apparent involvement of the lungs alone, those indicative of lung and lymph node involvement, and those apparently indicating lymphadenopathy alone. The pattern of the lung involvement is extremely variable, ranging from true miliary densities, through coarse nodulation and apparent linear fibrosis, to coalescent cirrhotic or pneumonic shadows, all with or without adenopathy. Findings in the individual case are not characteristic, but apparent excellent health in contrast with the extensive X-ray shadows may suggest the diagnosis. The miliary and nodular lesions are due to aggregations of sarcoids in the lung parenchyma. The linear lesions may be due to sarcoid lymphangitis, to lymph-œdema, to congestion, or, occasionally, to fibrotic changes. The intrathoracic lymph node enlargement tended to conform to a curious pattern in 13 of the 24 cases in which adenopathy occurred; there was simultaneous enlargement of both sets of hilar nodes and of only the right upper mediastinal or paratracheal nodes. The most frequent type of sarcoidosis encountered clinically is one with hilar lymphadenopathy and pulmonary infiltration. The possibility of sarcoidosis should be kept in mind during routine chest surveys, and it should be considered in the differential diagnosis of all cases of chronic painless lymphadenopathy, whether there are demonstrable lesions of the skin, bones and lungs or not. The diagnosis is not established by pathological examination alone, but only in conjunction with the appropriate clinical and bacteriological evidence to eliminate other granulomatous infections. A palpable lymph node or skin nodule or, failing these, an upper anterior mediastinal node should be obtained for microscopic examination. Absence of pulmonary involvement at one stage of sarcoidosis is no assurance that it will not be silently present months or years later. The course of a given case is usually quite unpredictable. The lesions may regress, remain stationary, or progress without apparent reason. This renders estimation of the beneficial effects of therapy, such as X radiation, extremely difficult.

Posterior Displacement of Lumbar Vertebræ.

ABRAHAM MELAMED AND DAVID J. ANSFIELD (*American Journal of Roentgenology*, September, 1947) consider that backward displacement of lumbar vertebræ, the fifth lumbar vertebra included, is a pathological entity which is encountered with sufficient frequency to deserve greater recognition. The following criteria of diagnosis are enumerated. (i) Degeneration with or without actual narrowing of the intervertebral space is essential. Radiological signs of disk degeneration are as follows: (*a*) narrowing of the intervertebral space; (*b*) reactive changes at articular margins of the vertebral segments; (*c*) vacuum phenomenon; (*d*) calcification of the intervertebral disk; (*e*) instability—abnormal relation of vertebræ, such as retrodisplacement, pseudo-spondylolisthesis *et cetera*; (*f*) abnormal motion of vertebræ; (*g*) alteration of the lumbar curve. (ii) The posterior border as well as the anterior border of the cephalad vertebral body must be displaced posterior to the corresponding portion of the caudad vertebra. The continuity of the lumbar curve is broken. (iii) Narrowing of the intervertebral foramina must be present—at least in the antero-posterior direction. If the intervertebral space is decreased, vertical narrowing of the intervertebral foramina will occur. The antero-posterior narrowing of these foramina results in an "hour-glass" appearance. This encroachment is due to approximation of the postero-inferior margin of the cephalad vertebral body and the superior articular process of the adjacent caudad vertebra. (iv) Displacement of the facets and/or widening of the apophyseal joint space must be present. If the facets are of the internal-external type, widening of the overlapping articular processes in the sagittal plane will be disclosed. If the facets face anteriorly and posteriorly, widening of the joint space must be demonstrated. (v) There is prominence or protrusion of the spinous process of the displaced vertebra on the sagit-

tal projection. (vi) The lumbar curve is altered. (vii) Radiological signs of retrodisplacement, not eliminated by technical means, are present.

Early Coccidioidomycosis.

H. W. JAMISON AND R. A. CARTER (*Radiology*, April, 1947) state that the diagnosis of coccidioidomycosis is not a difficult one if the physician is alert to the possibility of such a condition. The disease should be suspected in the case of any person recently returned from an endemic zone of infection, who presents the signs and symptoms of a respiratory infection. If a positive result to the coccidioidin test is obtained in these circumstances, a presumptive diagnosis of coccidioidomycosis is warranted. A negative result rules out the disease in all but the most severe infections. The diagnosis is established with certainty by a positive result to the complement-fixation or precipitin test or by recovery of the causative organism from the sputum in a culture or by guinea-pig inoculation. In the acute pneumonic phase of the disease the radiological appearance is non-specific, and differentiation from primary atypical pneumonia, from rheumatic pneumonitis and from other respiratory infections cannot ordinarily be made from the X-ray films alone. Residual "burned-out" nodular or cyst-like foci of coccidioidomycosis are quite characteristic in X-ray appearance and are seldom confused with other conditions when occurring in endemic areas. Among diseases to be differentiated are primary tuberculosis, metastatic carcinoma, congenital cyst, adult tuberculosis, lung abscess, and pyogenic and mycotic infections. The discrepancy between the clinical and radiological findings is often helpful in establishing the diagnosis. Cases in which mediastinal adenopathy is dominant are usually among the more severe and prolonged of infections and give rise to most of the fatalities. Among the conditions to be considered in the differential diagnosis are Hodgkin's disease, pulmonary tuberculosis, sarcoidosis, and bronchiogenic carcinoma.

PHYSICAL THERAPY.

Optimal Skin Tolerance Dose Levels.

B. JOLLES AND R. G. MITCHELL (*British Journal of Radiology*, October, 1947) report an investigation of the skin tolerance dose with the following physical factors: 180 kilovolts, 10 milliamperes of current, a filter of one millimetre of copper *plus* one millimetre of aluminium, 40 centimetres of focal skin distance, half value layer 1·25 millimetres of copper. The treatments were given three or four times a week. As it is believed that the tolerance depends in some way on the extent of untreated tissue surrounding the irradiated area, comparisons were made between treatment fields of the same area, only one of which had an elongation of three to one. The tolerance dose used as a standard of comparison was one causing a moist desquamation of the treated area, or in fields greater than 120 square centimetres of not less than the inner half of the treated area, healing with routine dressings in four weeks after the completion of treatment. Doses were given for periods of one, two, three, four, five or six weeks. An attempt was made also to estimate the tolerance dosage for a single treatment dose. The authors consider that in the X-ray reaction due to a single dose treatment (provided it is adequate) destruction prevails. In the protracted treatments the reaction is due to a mixture of destruction, repair and recovery. When a region of the body is treated by means of two adjoining fields the dose of tolerance must be decreased. The dose for the two adjoining fields should be calculated on the basis of the perimeter/area ratio and a table is given for the percentage reductions in the tolerance dose values from those permissible for single fields. The authors point out the limitations of the investigation, namely, that the site of application and the age and general condition of the patient have been neglected, and that only one set of factors were considered, namely, the area irradiated, its perimeter and the period of treatment. The experiments, however, are a definite step forward towards knowledge of optimal skin doses.

Cancer of the Eyelid.

HOWARD B. HUNT (*American Journal of Radiology*, February, 1947) states that ten to twenty years ago radium was preferred to X rays for treatment of cancer of the eyelid, but with more accurate dosimetry and improvement of apparatus X-ray therapy is now greatly superior to radium for treatment of these lesions. Cancer of the eyelid comprises from 9% to 17% of cancer of the skin affecting primarily the older age group. The clinical diagnosis involves a differentiation from benign neoplasms—benign moles, melanocarcinomata, hæmangiomata, seborrhœic warts and *xanthoma palpebrarum*—and inflammatory lesions including acute inflammations, *molluscum contagiosum*, chalazion and small indolent nodules on the margins which are not uncommon. As a result of irradiation of the eye, conjunctivitis and œdema of the lids usually occur about three to five days before the epidermitis of the skin; the cornea shows little reaction, as also do the iris and retina. The lens is very susceptible to injury by irradiation, although a period of three months to twelve years may occur before the cataract becomes apparent. The problem of post-irradiation cataract is of paramount importance in the treatment of cancer of the lid by either X rays or radium. The threshold dose productive of irradiation cataract does not appear to have been determined, but it has occurred after 875*r* delivered to the upper lid. A dose of 1100*r* to the lens produced a cataract within six months. Cataract in X-ray therapy of lesions of the lid can be prevented by eye shields of lead two millimetres in thickness and rubber covered on the corneal aspect; this thickness is not adequate protection against γ radiation. Additional precautions are to limit the treated area as much as possible to avoid scattering—the reduction of depth dose by employment of 50 kilovolts to 90 kilovolts with at most one millimetre of aluminium filter and short focal skin distance. With these precautions the risk of cataract is small. Other effects to be avoided are closure of the lachrymal duct, ectropion, entropion and trichiasis. Treatment given in the series of 100 cases was X-ray therapy in eighty cases, radium in three, radium and X rays combined in five, electro-surgery and X rays in seven, electro-surgery and radium in three, and electro-surgery alone in two cases. The conditions treated showed great variation in extent. The authors discuss methods of treatment and dosage used and also indicate the value of surgery.

Leuchæmia.

B. P. WIDMANN (*American Journal of Roentgenology*, April, 1946) states that deep X-ray therapy in chronic leuchæmia is generally acknowledged as an excellent palliative measure; the reports of results, however, show great variation. The author reports a series of 110 cases sufficiently severe to require admission of the patient to hospital and thus relatively comparable. Forty-nine patients were treated and followed and the results in this series are analysed. Of these 28 died and 21 were still receiving treatment. The average duration of life from the institution of treatment was 2·7 years in the myeloid group and 3·3 years in the lymphoid group. It is interesting to note that one patient lived for thirteen years, one for eighteen years and one for nineteen years; the author considers that these patients had relatively benign forms of the disease and the long life could not be attributed to irradiation. The blood count is a reliable criterion of the progress of the disease. Treatment in the author's opinion should be withheld until symptoms require alleviation. In this series many patients maintained good health with leucocyte counts varying from 50,000 to 700,000 per cubic millimetre. In these circumstances no irradiation was given unless there were symptoms of general indisposition, pain, or splenic or lymph gland enlargement. Patients with mild types of leuchæmia may carry on for years with little or no irradiation. Patients suffering from the fulminating type, with marked splenic or lymph gland enlargement and rapidly failing health, usually need frequent treatment. Review of the literature shows the great variations in technique with reference to size of field and region treated. Excellent results have been obtained with very different methods; no standard procedure of daily or total dose is uniformly satisfactory. The treatment must be varied according to the response of the patient. In general one should aim at finding the smallest dose which establishes an improvement in health. Usually 50*r* (in air) is a safe starting basis; this may be given according to the needs of the patient in frequency from daily to twice a month. A patient may become refractory to small doses after six months or so and an increase to 150*r* or 200*r* may be necessary. Identical results appear to be obtained with a current of 125 kilovolts and a filter of two to six millimetres of aluminium and with a current of 200 kilovolts and a filter of half a millimetre of copper and one millimetre of aluminium. The majority of patients were treated by fields of 15 by 15 centimetres over the spleen, mediastinum or ribs or directly to enlarged glands; general body irradiation was occasionally used. The author concludes that improvement in health and efficiency can be achieved in more than 50% of patients suitable for treatment and the life span is probably increased in a small percentage of cases.

Medico-Legal.

HOCKING VERSUS BELL.

On December 18, 1947, the Lords of the Judicial Committee of His Majesty's Privy Council delivered their judgement and allowed an appeal by Mrs. Stella Hocking, of Quirindi, New South Wales, against a judgement of the High Court of Australia. The High Court of Australia, whose judgements were published *in extenso* in THE MEDICAL JOURNAL OF AUSTRALIA of February 23, 1946, had dismissed an appeal by Mrs. Hocking against a judgement of the Full Court of New South Wales. The Privy Council's judgement was delivered by Viscount Simon and there were present at the hearing Lord Porter, Lord Uthwatt, Lord du Parcq and Lord Oaksey. The judgement is as follows.

This is an appeal by the plaintiff *in forma pauperis*, brought by leave of the Privy Council from a decision of the High Court of Australia. (Rich, Starke and McTiernan, JJ., Latham, C.J., and Dixon, J., dissenting.) The litigation out of which the appeal arises has a regrettably long history, for the case has been before a Judge and jury of New South Wales no less than four times. The jury on each occasion consisted of four members. After the trial, which resulted in a verdict and judgement for the plaintiff for £500, the Supreme Court of New South Wales ordered a new trial on the ground that the verdict was against the weight of evidence; at the second trial, and again at the third trial, the jury disagreed, being equally divided; and at the fourth trial (which is the one with which this appeal is immediately concerned) the jury found a verdict for the plaintiff for £800 damages, and the Trial Judge on January 21, 1944, gave judgement accordingly.

From this decision the defendant appealed to the Supreme Court of New South Wales, claiming that judgement should be entered for him or, alternatively, that a new trial should be ordered, and the appeal was heard by Davidson, Halse Rogers and Roper, JJ. Of the fourteen grounds adduced, the main one was that the verdict was against the weight of evidence and was such as no reasonable jury could have found. One of the further grounds was that, in the circumstances, the defendant was entitled "as a matter of law" to succeed. This ground is based on Section 7 of the Supreme Court Procedure Act, 1900, with the interpretation and application of which their Lordships will subsequently deal. The Supreme Court allowed the appeal and directed that judgement should be entered for the defendant, though Mr. Justice Roper's view was that the proper order to make would be for yet another trial.

The plaintiff then appealed to the High Court of Australia, which, by a majority, as already stated, affirmed the decision of the Supreme Court.

It is now necessary for their Lordships to state the nature of the claim and defence in this complicated case, and outline the evidence on either side. The whole of this evidence, which is very voluminous, has been closely scrutinized by the Board with the help of Counsel on either side, but their Lordships must emphasize that it is no part of their duty to express, or even to form, their own opinion on facts in controversy. A finding on such facts is for the jury. Their Lordships' function is to determine whether the verdict of the jury can be supported, and if not, whether the judgement for the defendant now appealed against can stand; if both of these questions were to be answered in the negative, then the question of a new trial would have to be considered. In these circumstances, it is not necessary for their Lordships to recount all the material evidence in detail, though they have considered every part of it with anxious care. For the purposes of the present appeal a summary of the salient matters is sufficient.

On January 17, 1941, the present appellant, Mrs. Hocking, issued a writ for damages for negligence against the present respondent, Dr. Bell, who is a surgeon of high standing and great experience, in respect of his treatment of her, following upon an operation of thyreoidectomy which he performed on her in Saint Luke's Hospital, Sydney, on March 15, 1938. No criticism is made of the skill with which the operation itself was performed, but what Mrs. Hocking alleges is that when, a few days later (in fact on March 17), Dr. Bell personally undertook the removal of the rubber drainage tube from her wound, he did not remove the whole of it, but negligently left *in situ* a portion of its inner end, which broke off—it is suggested that it was caught and held by a stitch—and never got it out. A few days later the wound, from whatever cause, became heavily infected. After many purulent discharges it ultimately closed at the end of June, and the plaintiff's case is that the foreign body, enclosed in a suppurating cavity, brought about violent and painful attacks of tetany, that is, uncontrollable spasms of the muscles. Such attacks might occur through infection interfering with the normal functioning of the parathyreoid glands. These attacks according to the plaintiff's case, recurred from time to time until a period of more than eighteen months from the operation had elapsed, and were accompanied by severe and painful swellings in the neck. But on October 2, 1939, during a particularly severe tetanic spasm, a portion of tube, as she alleges, was carried into her mouth, owing to the bursting out from her left tonsil of the abscess surrounding this foreign body. The plaintiff's case therefore essentially involves the view not only that a portion of the tube was left behind on March 17, 1938, but that it travelled from the thyreoid area into and through the tonsil. According to the plaintiff's evidence she could not do other than swallow this object, for her teeth were spasmodically clenched, and three days later she found it after a bowel motion. The object was not forthcoming at the trial, for it was, she said, owing to her weakness, accidentally dropped in the water-closet receptacle when she was emptying the commode-pan. It was carried away by the flush. But before this happened the plaintiff said that she had picked it out with her thumb and finger and examined it, and on the same day, after its loss, she made from recollection a pencil drawing of it—not, she said, to scale. The drawing was an exhibit at the trial. A particularly curious feature of the plaintiff's description and drawing is that there projected from the recovered piece of tube two filaments which had the appearance of wires, and that in the exposed interior, where the tube appeared to have a "V"-shaped cut, there appeared something which she described as like "a marine sponge" or "swab". After the alleged expulsion of this object the appellant never had any further attack of tetany.

To this remarkable story, the respondent, besides criticizing it by reference to hospital records, and by what had been said at the earlier trials, opposed an impressive body of scientific evidence drawn from medical experts of high qualifications and experience, to the effect that the alleged travelling of a foreign body, and especially such a body as the plaintiff alleged, from thyreoid to tonsil could not in fact occur. According to this evidence, the contents of that portion of the neck are too closely packed, and the compartments of the neck too completely separated, to permit of such passage; moreover, the suppuration involved in such an abscess eating its way by any route that could be suggested, between these two points, must, according to this evidence, in any case have involved vital organs with fatal results. In a word, the respondent's case was that the thing was impossible, and therefore that it did not happen. A contrary view was taken by two medical experts called by the plaintiff, one, Professor Welsh, a former Professor of Pathology in the University of Sydney, and the other, Dr. Thompson, and much depends in this appeal on examining the testimony of these two witnesses in order to see whether in the result they conceded the essential proposition of the defendant's experts or whether there emerged at the end a difference of opinion as to the possibility of the events alleged by the plaintiff having happened.

At the trial the Learned Judge, in the course of a careful summing up, which showed a full appreciation of the difficulties in the plaintiff's way, invited the jury to say whether it accepted the plaintiff's account of the object said to have been left in the wound after the operation, and he provided each member of the jury with a specific question on the point, which the jury, as the Learned Judge pointed out, was not obliged to answer. The jury, however, did answer it in a modified form, in addition to finding a verdict for the plaintiff. Their Lordships take the view that the two answers of the jury must be combined, and the verdict would therefore run as follows:

> We find that the defendant left in the site of the operation a piece of rubber tube of a length somewhat less than two inches, cut off straight at one end, and torn at the other, part of which tube had been cut down one side and from which protruded some material which looked like wire and a swab from the torn end of the tube, and we accordingly find a verdict for the plaintiff for £800.

Some discussion arose before their Lordships and at the earlier stages of the trial as to the meaning to be attributed to the phrase "which looked like wire and a swab", but their Lordships have no doubt that the true meaning is "which looked like wire and looked like a swab"—there is no finding that either of the things referred to was in fact wire or in fact a swab. The evidence of the plaintiff when fairly read

is, in their Lordships' opinion, plainly asserting not the true character of these strange objects, but merely what they looked like. If the plaintiff's story were to be accepted, it would not be inconsistent with her description to imagine that the filaments were pieces of stitching material (if indeed stitching material of this length and durability were used), and that the thing like a swab was detritus of some sort picked up in the passage of the foreign body at some stage in its course. The real issue in the case, in their Lordships' opinion, does not turn on those minutiæ, strange and surprising as they may be, but upon the broad issue whether the jury should be upheld in believing the plaintiff's story in view of the mass of evidence brought against its possibility.

In dealing with appeals in New South Wales, there is no provision corresponding to Order 58, Rule 4, of the Rules of the Supreme Court in this country, by which the appeal tribunal is authorized to draw inferences of fact and to enter judgement if it thinks fit notwithstanding the verdict of the jury. In making this observation their Lordships must not be understood to imply that, if such a rule existed in New South Wales, the present case might fall within it, for the Supreme Court of New South Wales, in directing that, notwithstanding the verdict of the jury, judgement should be entered for the defendant, was not merely drawing an inference of fact, but was taking the view that no reasonable jury could find otherwise than against the plaintiff. The observations of Chief Justice Latham on this point in the course of his judgement in this case were not, as their Lordships understand, disputed by the defendant's counsel, and in any case their Lordships consider that these observations were perfectly correct. The passage is as follows:

> After a trial by jury, the Full Court of New South Wales upon appeal has no power to draw inferences of fact; and though it may order a new trial where the verdict is against evidence and the weight of evidence, it cannot order a verdict to be set aside and judgement to be entered for the party against whom the verdict was given unless the conditions prescribed by the Supreme Court Procedure Act, 1900, Section 7, are satisfied. Section 7 provides that:
>
> > In any action, if the Court in Banco is of opinion that the plaintiff should have been non-suited, or that upon the evidence the plaintiff or the defendant is as a matter of law entitled to a verdict in the action or upon any issue therein, the Court may order a non-suit or such verdict to be entered.
>
> Thus in the present case the Full Court could properly order a verdict to be entered for the defendant only if the defendant is "as a matter of law entitled to a verdict". If there is evidence upon which a jury could reasonably find for the plaintiff, unless that evidence is so negligible in character as to amount to a scintilla, the judge should not direct the jury to find a verdict for the defendant, nor should the Full Court direct the entry of such a verdict. The principle upon which the section is based is that it is for the jury to decide all questions of fact, and therefore to determine which witnesses should be believed in case of a conflict of testimony. But there must be a real issue of fact to be decided, and if the evidence is all one way, so that only one conclusion can be said to be reasonable, there is no function left for the jury to perform, so that the Court may properly take the matter into its own hands as being a matter of law, and direct a verdict to be entered in accordance with the only evidence which is really presented in the case.

The Chief Justice's application of the section is, as it seems to their Lordships, perfectly in point. If, at the end of the hearing of witnesses, the evidence is all one way, so that no jury can reasonably find for the plaintiff, and a verdict and judgement in favour of the plaintiff are nevertheless given, it is within the competence of the Supreme Court to direct that verdict and judgement should be entered for the defendant. The main question in this appeal really is whether that is the situation with which the Supreme Court had to deal.

It appears to their Lordships that the situation that there is no evidence upon which a jury can reasonably base their verdict may arise at one of two stages, either at the end of the plaintiff's case or, sometimes, at the close of all the evidence. When the burden of proof rests on the plaintiff and at the end of the plaintiff's case this burden has not been discharged, the plaintiff may at that stage be non-suited: mere speculative possibility not being the same thing as *prima facie* proof. It would not, in their Lordships' opinion, be possible to say in the present case that when the plaintiff's case was closed there was no evidence to support it. To go no further, there was the evidence of the plaintiff herself, as well as that of her two medical witnesses. But there is a second stage at which it may sometimes be correct to decide that the plaintiff cannot succeed and that the defendant is entitled to judgement. This is at the end of all the evidence if there is undisputed evidence of further facts called by the defendant which render it impossible to accept the plaintiff's story, or which negative the assumption on which the plaintiff's case depends. To give a simple example, if a plaintiff brings an action for trespass and proves that the defendant, without his permission, entered upon his premises, this situation established at the end of the plaintiff's case would not justify a non-suit; but if the defendant thereupon proves beyond dispute that he was authorized by lawful authority to enter, the plaintiff's case collapses and there is nothing left in issue upon which the jury can decide in the plaintiff's favour. This is the essence of the defendant's contention here. He claims that though the plaintiff made a *prima facie* case in the first instance, the evidence subsequently called established beyond dispute that what the plaintiff alleged and swore to have happened could not possibly have happened, and, this not being an age of miracles, the defendant must succeed. Their Lordships must therefore proceed to examine the testimony for the purpose of seeing whether, on the evidence taken as a whole, this is the resultant position.

On this crucial question their Lordships find themselves in substantial agreement with the judgements of Chief Justice Latham and Mr. Justice Dixon. Whilst not expressing any opinion as to what their own view would have been if the responsibility of deciding the facts rested with them and not with the jury, who saw and heard the witnesses, they agree with Chief Justice Latham and Mr. Justice Dixon that there was evidence upon which the jury were entitled to find a verdict in favour of the plaintiff. It cannot be suggested, nor has it been suggested, that any matter of fact has not been fully and sufficiently sifted. There was a conflict of evidence between the witnesses for the plaintiff and the witnesses for the defence on nearly all the material issues in the case, namely, the circumstances of the removal of the tube by the defendant on March 17, 1938, the condition of the plaintiff's health from 1938 to October, 1939, the condition of the plaintiff's left tonsil after October 2, 1939, and the possibility of the object described by the jury travelling from the thyreoid gland to the tonsil.

In their Lordships' view it is impossible to say that there was not evidence for the plaintiff which entitled the jury to resolve all these issues in favour of the plaintiff. In particular, there was the evidence of the plaintiff as to the removal of the tube and also the evidence of Mrs. Warburton which may have influenced the jury on this question; there was the evidence of the plaintiff, of her husband, of Sister Sly, of Mr. and Mrs. Nancarrow, of Fisher, of the hospital records, of Dr. O'Hanlon's contemporary letters as to the plaintiff's condition coupled with the admitted fact that the plaintiff was throughout treated for real tetany and that the diagnosis of hysteria was never suggested by anyone at the time except by Dr. Ritchie to the defendant and by Dr. O'Hanlon in one sentence of the letter of January 17, 1939; there was the evidence of Professor Welsh and Dr. Thompson that the plaintiff's left tonsil when examined during the first trial was in a condition consistent with the passage of the tube through it; and there was the evidence of these two doctors that, in their opinion, an object such as was described by the jury might have travelled from the thyreoid gland to the left tonsil.

It is true that Professor Welsh and Dr. Thompson agreed that the construction built up at a previous trial when the plaintiff was in the box which was produced as exhibit "P" and which had actual wires in it, could not have travelled from the thyreoid gland to the tonsil, but the question put to the jury and their answer to it, in their Lordships' view, was not intended to refer to exhibit "P". The plaintiff more than once repudiated the suggestion that "P" was an exact replica of the actual object.

If, therefore, the jury believed the evidence of the plaintiff as to her experiences from October 2 to October 5, 1939—as to which there was not, and, of course, could not be, contradictory evidence—they were entitled to draw the inference from the body of evidence to which their Lordships have referred that an object such as the jury described had been left by the defendant in the plaintiff's neck after the operation. The evidence called for the defence was, no doubt, entitled to great weight and the circumstances of the case are of a most unusual nature; but their Lordships do not think, particularly in view of the results of the

four trials, that it can be said that no reasonable jury could have reached the verdict at which the jury in this case arrived. There is therefore no adequate ground for ordering a new trial.

Their Lordships will therefore humbly advise His Majesty that this appeal shall be allowed and judgement entered for the plaintiff for £800 with such costs here as are allowed to persons appearing *in forma pauperis* and taxed costs in the courts of Australia.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held at the Adelaide Children's Hospital on October 30, 1947, DR. A. BRITTEN JONES in the chair. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital. Part of this report appeared in the issue of February 14, 1948.

Probable Neuroblastoma of the Adrenal Gland.

DR. ERIC SIMS showed an eight months old baby suffering from a probable neuroblastoma of the adrenal gland with secondary deposits in the liver and in the skull around the orbits. The child had first been seen at the age of six months when her mother had taken her to Dr. Malcolm Cockburn on account of vague ill health of two weeks' duration. Routine examination revealed a large liver and (it was thought) spleen; and she was admitted to the Children's Hospital for investigation with a possible diagnosis of leuchæmia. The blood picture, the bone marrow, and X-ray pictures of the long bones were, however, normal. After several weeks in hospital a hæmorrhagic streak was noticed in one eyelid, and the liver was then observed to be definitely irregular on palpation of the abdomen. Since then, bony-hard lumps had appeared at the margins of the orbits, and the eyes had became proptosed. A hard lump was also palpable in the left ramus of the mandible. These deposits had not been satisfactorily demonstrable in X-ray photographs of the skull, but in one slightly oblique view new bone formation in the mandibular deposit could be seen. The mass in the left side of the abdomen was now also irregular, and could conceivably be arising from the left adrenal region.

Dr. Sims commented that neuroblastomata could also arise from other chromaffin tissue in the abdomen and elsewhere. The tumour was peculiar to infancy and childhood, and the secondary deposits were often a more prominent feature than the primary growth which, on occasions, had to be searched for microscopically. The tumour consisted of small round cells (primitive sympathetic neuroblasts) with a tendency to "rosette" formation around bundles of fibrils.

Dr. Sims said that in two other cases of neuroblastoma at the Adelaide Children's Hospital that year (confirmed by autopsy) severe hæmorrhage had occurred into the various deposits. He showed X-ray photographs of one of the patients which illustrated the tendency to the occurrence of multiple deposits in most of the long bones as well as in the flat bones of the body. The differential diagnosis of such a condition usually was between neuroblastoma and Ewing's tumour. The bone deposits caused both erosion and the formation of periosteal new bone and, in the skull, could lead to a forest of spicules projecting into the cranial cavity from the inner table. In such cases evidence of increased intracranial pressure was also present. The textbook differentiation of the Hutchinson type (with metastases in the skull) and Pepper type (with metastases in the liver) was not now generally supported. Mixed types such as in the patient shown were not unusual.

Discussing treatment, Dr. Sims said that successful treatment by surgery and irradiation had been reported in a number of cases, even after metastasis to the liver, but apparently no patients had recovered after skeletal involvement, and no treatment was contemplated for the child under discussion. He remarked, in conclusion, that perhaps the autopsy findings would be available for the next meeting.

Tuberculous Meningitis Treated with Streptomycin.

DR. F. N. LE MESSURIER showed a male infant, aged eleven weeks, who had been admitted to the Adelaide Children's Hospital on May 3, 1947, with the history of coughing paroxysms for one month. General examination revealed no physical signs. The child's weight was eleven pounds seven ounces, whereas the birth weight had been six pounds three ounces. During the next few days the child had had several asthma-like attacks, which were relieved by adrenaline. On May 5 the result of a Mantoux test (1 in 1000) was negative, whereas X-ray examination of the chest on May 7 suggested some degree of atelectasis of the middle lobe of the right lung. The condition had remained stationary for one month, with recurrent wheezes, and some inspiratory rib retraction. X-ray examination of the chest was repeated on June 3, and revealed a rounded shadow projecting from the right hilar region. The Mantoux test was now repeated and the result found to be positive. The mother was sent to the chest clinic at the Adelaide Hospital, where she was found to have tubercle bacilli in the sputum. A further X-ray examination of the chest on July 26 revealed signs suggestive of miliary spread. On July 28 the anterior fontanelle was found to be bulging and there was slight neck rigidity. A lumbar puncture revealed cerebro-spinal fluid under pressure; the fluid contained 157 cells per cubic millimetre, of which 80% were polymorphonuclear leucocytes and 20% lymphocytes, 97 milligrammes of protein *per centum* and 700 milligrammes of chlorides *per centum*; but no tubercle bacilli were seen.

Streptomycin, 240 milligrammes given intramuscularly each day (30 milligrammes every three hours), had been commenced on July 28 and continued to the date of the meeting. The total dosage had been 20·76 grammes. Intrathecal injection of 100 milligrammes daily had been commenced on July 29 and continued (with the omission of Sundays) until September 9, a total amount of 3·66 grammes in 37 injections.

Examination of the cerebro-spinal fluid, whilst the patient was receiving intrathecal injections, had shown that the white cells fluctuated widely and erratically between the minimum of 270 and a maximum of 3000 per cubic millimetre and were predominantly polymorphonuclear leucocytes. The protein content generally ranged between 100 and 200 milligrammes *per centum*; on one occasion it reached 275 milligrammes *per centum* (August 21). The chloride content ranged between 700 and 620 milligrammes per *centum*. After the intrathecal administration of streptomycin had been discontinued, the cells dropped in number and changed predominantly to lymphocytes. The baby's rectal temperature had hovered between 99° and 100° F., from the time of admission until July 22, when it gradually commenced to swing, reaching 102·4° F. on July 28, the day on which meningitis was diagnosed. The temperature continued to swing to 102° or 103° F. each day for the next five weeks. During the sixth week after commencement of treatment the evening temperature was usually 101° F.; after the intrathecal administration streptomycin was stopped, the fever slowly subsided to the usual daily maximum of 99° to 100° F. by the end of September (nine weeks after commencement of treatment).

On August 22 the child had commenced having attacks of convulsive twitching, mainly affecting the eyelids, face and arms. Those attacks recurred intermittently until the intrathecal injections were stopped eighteen days later. At times he was rather cyanosed and in some respiratory distress, with inspiratory wheezes, but he usually sucked a bottle well, taking five to seven ounces of full lactone syrup milk with baked flour per feed without vomiting. He was, at the time of the meeting, very bright and well, although still showing evidence of some pressure on the trachea by the mediastinal glands. A further X-ray examination carried out on October 29 had shown that the miliary "snow storm" had apparently resolved. On October 30 examination of the cerebro-spinal fluid revealed 40 milligrammes *per centum* of protein and 720 milligrammes *per centum* of chlorides, and the cell count was 15 lymphocytes per cubic millimetre.

Dr. Le Messurier remarked that the baby clinically showed some tuberculous infection of the meninges, although tubercle bacilli were not found in the cerebro-spinal fluid, and he had made definite progress under streptomycin. It was too early to report a cure, but it must be agreed that, without streptomycin, the baby would have died some weeks before.

DR. ERIC SIMS said that four patients with tuberculosis had been so treated at the Adelaide Children's Hospital that year. The first child (who had been shown at the British Medical Association clinical meeting in April) had survived ninety-four days before succumbing. The case had been fully reported in THE MEDICAL JOURNAL OF AUSTRALIA in July. One other child, aged seven years, had survived for forty days, but at no stage showed any very hopeful response to the drug. The baby shown at the meeting was the first one so far to survive and manifest a return of the cerebrospinal fluid to a more or less normal state As was the

general experience, the cell count and protein content had fluctuated widely and erratically during the intrathecal administration of streptomycin, but at the time of the meeting, seven weeks after administration by that route had been discontinued, the baby was alert and well, and the cerebro-spinal fluid contained only 15 cells per cubic millimetre and 40 milligrammes *per centum* of protein. The miliary "snowstorm" in the X-ray film of the lung was also clearing in a gratifying manner. It was fully realized that the tubercle bacillus had not been isolated and therefore final proof of diagnosis was lacking; but the march of events was sufficiently typical to convince most reasonable people. The intramuscular injection of streptomycin was still being continued every three hours in a total dosage of 20 milligrammes per pound of body weight per day. That scale followed the precept of some American writers and had the advantage of economy when one was dealing with a baby. The baby under discussion, of course, still had his enlarged tracheo-bronchial glands which occasionally caused respiratory distress. The prognosis presumably was still in doubt, but that one attack of miliary spread and meningitis was probably overcome.

Dr. Sims went on to say that the fourth patient under treatment was also a baby, aged four months, when first seen. Treatment had been commenced for the primary pulmonary focus (again due to contact with an infectious mother) before any miliary spread occurred. The baby was still very well after eight weeks of treatment, but had developed numerous discharging tuberculous abscesses in the neck. It was considered justifiable to commence streptomycin treatment of babies with primary pulmonary tuberculosis if possible before meningitis had supervened, and a fifth patient in that category was, in fact, about to commence on a course of the drug.

Congenital Obstruction of the Urethra.

DR. L. A. WILSON showed a boy, aged ten weeks, who had had difficulty in passing urine since birth. At the age of seven weeks he had become very distended and a large mass was felt in the lower part of the abdomen just to the right of the mid-line. The condition was at first thought to be Hirschsprung's disease, but X-ray examination with and without the administration of a barium enema did not reveal any dilatation of the bowel. After the child had not passed any urine for two days, a soft catheter was passed and six ounces of infected urine were withdrawn. The size of the abdominal tumour diminished, but it was still palpable. Masses could be felt in both loins. A cystogram revealed enormous ureters, but no dye was visible in the kidney area. Post-mortem examination had revealed grossly dilated ureters, a small bladder, and dilatation of the proximal part of the urethra with Proust's valves in the region of the verumontanum.

An X-ray film was shown of a similar condition in a boy, aged eight years, who had been operated on by Dr. Wilson some years previously; this showed the contrast medium in the dilated proximal part of the urethra, the bladder, the ureters and the kidneys.

DR. G. H. BURNELL said that Dr. Wilson had asked why the bladder did not dilate in the patient presented by him; the answer was that the child had not lived long enough for that to happen, but it would inevitably have happened had the child survived. Dr. Burnell was interested to hear Dr. Wilson stress the importance of the dilated state of the prostatic part of the urethra in the diagnosis of the condition of congenital valvular obstruction of the posterior urethra; that had first been pointed out by Young, and Dr. Burnell had shown a patient, about 1930, on whom he had operated, and in whom it had been possible for him to get his finger well down into the prostatic part of the urethra, to open the urethra onto his finger, and then to excise the valves under direct vision. Young was able to do that *per urethram* with a small resectoscope, but such small instruments were not available locally. Dr. Burnell had performed one such operation *per urethram*, but that was in a patient who had survived to the age of forty years.

Multiple Intussusceptions in One Family.

Dr. Wilson then showed a boy, aged six years, on whom he had operated for an intussusception in August, 1947. That was the boy's fourth attack. The first attack had occurred when the boy was aged one year and ten months and had been cured by operation. The condition had recurred one month later and again operation was performed. When five years old the boy had had a third intussusception which was reduced hydrostatically. No attempt was made at any operation to fix any part of the ileum or caecum. At the last operation there were many firm adhesions between the caecum and the abdominal wall; these apparently did not prevent the intussusception.

Dr. Wilson remarked that as was usual in the recurrent attacks the mother had made a diagnosis before admission. Three sisters had had intussusceptions at the age of six months. Dr. Wilson said that he hoped to publish further details later.

The Effect of Thiouracil on the Unborn Child.

DR. IVAN MAGAREY presented a report of a case which illustrated the effect on the unborn child of thiouracil administered to the mother. The case will be reported in full in a subsequent issue of the journal.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 26, of February 5, 1948.

AUSTRALIAN MILITARY FORCES.

Reserve of Officers.

Australian Army Medical Corps.

The following officers are transferred to the Reserve of Officers of the Military Districts shown on the dates indicated. Officers holding temporary rank relinquish such temporary rank with effect from the date of transfer to the Reserve of Officers:

Captains QX53971 D. R. L. Hart (1st Military District), 16th December, 1947, and QX53968 D. A. Henderson (1st Military District), 26th November, 1947.

70th Camp Hospital.—NX208068 Captain J. Cameron (2nd Military District), 13th December, 1947.

Retired List.

Australian Army Medical Corps.

3rd Military District.—Lieutenant J. I. Stanwell (*née* Freeman) is placed upon the Retired List (3rd Military District), with permission to retain her rank and wear the prescribed uniform, 16th December, 1947.

Reserve Citizen Military Forces.

Australian Army Medical Corps.

3rd Military District.—Lieutenant R. E. Matthews is placed upon the Retired List (3rd Military District) with permission to retain her rank and wear the prescribed uniform, 13th September, 1946 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 234 of 1946, promulgated in *Commonwealth Gazette* No. 215 of 1946).

Interim Army.

Australian Army Medical Corps.

VX2 Major-General S. R. Burston, C.B., C.B.E., D.S.O., V.D., relinquishes the appointment of Director-General of Medical Services, Adjutant-General's Branch, Army Headquarters, and is placed upon the Retired List (3rd Military District) with permission to retain his rank and wear the prescribed uniform, 7th January, 1948.—(Ex. Min. No. 18—Approved 30th January, 1948.)

Post-Graduate Work.

POST-GRADUATE AFTERNOONS AT PRINCE HENRY'S HOSPITAL, MELBOURNE.

UNDER the auspices of the Royal Australasian College of Surgeons, each Friday afternoon from February 6 to November 26, 1948, inclusive, will be devoted to post-graduate education in surgery at Prince Henry's Hospital, Melbourne. All medical graduates are eligible to attend, and those desiring to do so must register with the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne. At 2 p.m. on each Friday a demonstration of pathological specimens will be given by Dr. J. D. Hicks, Director of Pathology at Prince Henry's Hospital. The following

syllabus of lectures and operative demonstrations, beginning at 2.30 p.m., has been arranged:

February 20, Dr. J. G. W. Ashton, "Paralytic Ileus and Fluid Balance"; February 27, Dr. R. G. Worcester, "Ovarian Tumours".

March 5, Dr. L. Doyle, "Gastrectomy for Ulcer"; March 12, Dr. L. Doyle, "Gastrectomy for Carcinoma"; March 26, Dr. L. Doyle, "Thyreoidectomy".

April 2, Dr. Hamley Wilson, "Empyema"; April 9, Dr. W. B. Wishart, "Vesical Malignancy"; April 16, Dr. A. J. W. Ahern, "Bursæ and Tendon Sheaths"; April 23, Dr. D. Donald, "Inguinal Hernia"; April 30, Dr. A. Joyce, "Eye Cases", and Dr. Hamley Wilson, "Diaphragmatic Hernia".

May 7, Dr. L. Doyle, "Carcinoma of the Breast"; May 14, Dr. L. Doyle, "Cholecystectomy"; May 21, Dr. L. Doyle, "Block Dissection of Glands of the Neck (Local Anæsthesia)"; May 28, Dr. L. Doyle, "Colonic Carcinoma".

June 4, Dr. T. O. Sayle, "Fistula in Ano"; June 11, Dr. J. Jens, "Internal Derangement of the Knee Joint"; June 18, Dr. E. Harbison, "Injuries to the Hand"; June 25, Dr. W. B. Wishart, "Genito-Urinary Tuberculosis".

July 2 and 9, Dr. W. A. Hailes, "Surgery of Hernia"; July 16, Dr. W. A. Hailes, "Carcinoma of the Breast"; July 23 and 30, Dr. W. A. Hailes, "Intestinal Anastomosis".

August 6, Dr. F. D. Burke, "Cysts in the Neck"; August 13, "Anæsthetic Methods" (lecturer to be announced), and Dr. S. Williams, "Chemotherapy"; August 20, Dr. S. Reid, "Vascular Surgery"; August 27, Dr. V. Stone, "Breast Malignancy".

September 3 and 10, Dr. W. A. Hailes, "Surgery of the Gall-Bladder and Biliary Tract"; September 17, Dr. W. A. Hailes, "Amputation Stumps"; September 24, Dr. W. A. Hailes, "Malignant Glands of the Neck".

October 1, to be announced; October 8, Dr. R. G. Worcester, "Carcinoma of the Vulva"; October 15, Dr. A. J. W. Ahern and Dr. D. Donald, "Pilonidal Sinus"; October 22, Dr. K. Hallam, demonstration of X-ray films and operative demonstration, demonstrator to be announced; October 29, ear, nose and throat demonstration, and operative demonstration, demonstrators to be announced.

November 5, Dr. C. W. B. Littlejohn, "Painful Back"; November 12, Dr. C. W. B. Littlejohn, "Painful Foot"; November 19, Dr. C. W. B. Littlejohn, "Painful Shoulder"; November 26, Dr. C. W. B. Littlejohn, "Painful Wrist".

COURSE IN MEDICINE FOR M.R.A.C.P. AND M.D. PART II, 1948.

THE Melbourne Permanent Post-Graduate Committee announces the following course in thoracic diseases, suitable for candidates for the M.R.A.C.P. and for Part II of the M.D. The course will be under the direction of Dr. Clive Fitts, and will take place in March and April, 1948, at 2 p.m. on the days specified:

March 16 (Tuesday): "Anatomy of the Bronchial Tree", Dr. T. H. Steel, Alfred Hospital. March 18 (Thursday): "Radiology of the Lungs in Relation to Clinical Medicine", Dr. C. Fitts, Royal Melbourne Hospital. March 23 (Tuesday), "Aspects of Pulmonary Tuberculosis", Austin Hospital. March 25 (Thursday): "Aspects of Pulmonary Tuberculosis", Gresswell Sanatorium. March 30 (Tuesday): "Surgical and Medical Aspects of Non-Tuberculous Diseases of the Lung", Mr. C. J. O. Brown and Dr. Eric Clarke, Alfred Hospital. April 1 (Thursday): "Industrial Diseases of the Lungs", Dr. W. J. Newling, Saint Vincent's Hospital.

The fee for this course is £3 3s. Applications for enrolment, accompanied by the fee, should be made with the Secretary of the Committee, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1, not later than two weeks before the start of the course. Those accepted for the Commonwealth Reconstruction Training Scheme in 1948 may attend free of charge.

NUFFIELD MEDICAL FELLOWSHIPS.

THE Nuffield Foundation proposes to award annually a number of travelling fellowships to enable Australian graduates to gain in the United Kingdom training and experience to fit them for medical teaching and research in Australia. The fellowships are open to both men and women, preferably between twenty-five and thirty years of age, and will normally be tenable for one year. Their value will be from £600 to £800 sterling, according to the candidates' needs, *plus* travelling expenses.

Applications for fellowships to be awarded this year should be lodged by March 31, 1948, with the Secretary, Nuffield Foundation Australian Advisory Committee, c.o. the University of Melbourne, from whom application forms are available.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

SEMINAR IN MEDICAL STATISTICS.

THE Post-graduate Committee in Medicine in the University of Sydney announces that a seminar on the "Valuation of Scientific Records in Hospitals" will be conducted by Dr. H. O. Lancaster on Wednesday, March 10, 1948, at 5.45 p.m. at the School of Public Health and Tropical Medicine, University Grounds.

Correspondence.

PROMINENCE OF THE EYES IN FATHER AND DAUGHTER.

SIR: Dr. Joske's Mr. H. (January 17, 1948) is, I think, an individual who used to rouse my technical curiosity when he made his appearance in a neighbouring town where I practised thirteen years ago. At that time I considered him to have a moderate degree of oxycephaly; a profile view would show his "steeple head".

Sheldon's "Diseases of Infancy and Childhood" says "as a rule only one member of a family is affected, but hereditary and familial instances have been recorded"; but a local authority advises me that neither oxycephaly nor buphthalmos are regarded as genetically transmissible conditions.

Yours, etc.,
C. T. PIPER.

Shell House,
North Terrace,
Adelaide.
January 30, 1948.

A BASIC ROUTINE FOR POST-OPERATIVE TREATMENT AFTER LAPAROTOMY.

SIR: My friend, Mr. Leo Doyle, in his article on post-operative treatment, has upset my ideas on absorption from the alimentary tract by his statement that there is almost no absorption of water in the small intestine.

I cannot understand how bile salts and cholesterol and substances taken by mouth can be absorbed from the small intestine, except in dilute solution, that is, in the presence of a much greater mass of water. Nor can I understand the marked diuresis which occurs within thirty minutes of taking one and a half litres of water by mouth on an empty stomach. Moreover, I have noticed neither excessive thirst nor dehydration in my patients with ileostomy. Nor have these patients complained that water taken slowly by mouth passes out through the fistula.

I would therefore be grateful to Mr. Doyle if he would set out the evidence upon which his statement is based.

Yours, etc.,
V. J. KINSELLA.

235, Macquarie Street,
Sydney,
February 7, 1948.

THE TREATMENT OF PSYCHOSES AND PSYCHONEUROSES BY ELECTROPLEXY (ELECTRIC SHOCK THERAPY) IN A GENERAL HOSPITAL.

SIR: In thanking Professor Bostock and Dr. Phillips for their paper on electroplexy which is one of the best reviews of the subject I have read, I think it is a pity, having regard to the recent violent attack upon this form of treatment by Dr. Winnicott, of London, that the authors did not express their opinion more forcibly on the value of the treatment. They summarize their findings as that electroplexy is "of

considerable use" in anxiety states. This vague statement is based on some weird findings; for example, from six months to a year after discharge from hospital a follow-up investigation was made. Of the 50 anxiety states, 19 were traced. Of the 19, 13 were well and working, 14 claimed to have benefited and five denied benefit. Is there something wrong with this arithmetic? The same observation applies to the statement on depressive states; 22 were traced out of 50, 14 were well and working, 15 said they had benefited, seven said they derived no benefit.

With one part of the paper I find myself compelled boldly to disagree emphatically, and that is that the treatment can be used for "diagnostic-curative" purposes by the fact that some patients after a few treatments "revealed" a fairly severe psychosis. The authors offer no evidence at all to justify the use of the word "revealed" and no evidence to show why the word "caused" should not be used instead of "revealed".

Yours, etc.,
John A. McCluskie.

Department of Repatriation,
Box F352, G.P.O.,
Perth.
January 28, 1948.

Medical Prizes.

THE STEWART MacARTHUR PRIZE.

The Medico-Legal Society of Victoria is this year awarding the Stewart MacArthur Prize of £25 which is open to members of the legal and medical professions. The closing date for entries for this award is April 30, 1948. The conditions are as follows.

1. The prize shall be of twenty-five pounds and shall be open for competition in the year 1940 and thereafter in each alternate year.
2. The prize shall be open to undergraduates taking the legal or medical course at the University of Melbourne, and articled law clerks, and to legally qualified medical or legal practitioners resident in Victoria of not more than three years' standing.
3. Each essay submitted shall be upon a subject of medico-legal interest which shall be selected by the candidate.
4. An essay in collaboration between a medical and a legal candidate may be submitted, and in the event of such an essay being awarded the prize, the prize shall be divided between the authors.
5. All essays submitted shall become the property of the Medico-Legal Society of Victoria which may, at the discretion of the committee, publish any of them in the *Proceedings* of the Society or otherwise deal with them as the committee may think fit.
6. The committee of the Medico-Legal Society of Victoria shall appoint examiners and the award will be made on their recommendation in the month of June of alternate years.
7. The closing date for entries shall be April 30 in the year in which the prize is to be awarded.
8. The prize shall not be awarded if either the examiners or the committee of the society think that the standard of the essay or essays submitted is not sufficiently high to justify the award of the prize.

Research.

THE ELLA SACHS PLOTZ FOUNDATION FOR THE ADVANCEMENT OF SCIENTIFIC INVESTIGATION.

During the twenty-fourth year of the Ella Sachs Plotz Foundation for the Advancement of Scientific Investigation, fifty-two applications for grants were received by the trustees, twenty-one of which came from the United States, the other thirty-one coming from thirteen different countries in Europe, Asia and North and South America. In the twenty-four years of its existence the Foundation has made five hundred and seventy-six grants which have been distributed to scientists throughout the world.

In their first statement regarding the purposes for which the fund would be used, the trustees expressed themselves as follows:

1. For the present, researches will be favoured that are directed towards the solution of problems in medicine and surgery or in branches of science bearing on medicine and surgery.
2. As a rule preference will be given to researches on a single problem or on closely allied problems; it is hoped that investigators in this and in other countries may be found whose work on similar or related problems may be assisted so that more rapid progress may be made possible.
3. Grants may be used for the purchase of apparatus and supplies that are needed for special investigations and for the payment of unusual expenses incident to such investigations, including technical assistance, but not for providing apparatus or materials which are ordinarily a part of laboratory equipment. Stipends for the support of investigators will be granted only under exceptional circumstances.

In the past few years the policy outlined in paragraph 2 has been neglected and grants will be given in the sciences closely related to medicine without reference to special fields. The maximum size of grants will usually be less than $500.

Applications for grants to be held during the year 1948-1949 must be in the hands of the Executive Committee before April 15, 1948. There are no formal application blanks, but letters asking for aid must state definitely the qualifications of the investigator, an accurate description of the research, the size of the grant requested and the specific use of the money to be expended. In their requests for aid, applicants should state whether or not they have approached other foundations for financial assistance and what other sources of support are relied on for research. It is highly desirable to include letters of recommendation from the directors of the departments in which the work is to be done. Only applicants complying with the above conditions will be considered.

Applications should be sent to Dr. Joseph C. Aub, Massachusetts General Hospital, Fruit Street, Boston 14, Massachusetts, United States of America.

Obituary.

GERALD DENIS KENNA.

We regret to announce the death of Dr. Gerald Denis Kenna, which occurred on February 7, 1948, at Albury, New South Wales.

HERBERT RICHARD LETCHER.

We regret to announce the death of Dr. Herbert Richard Letcher, which occurred on February 8, 1948, at Adelaide.

DEVEREUX GWYNNE-HUGHES.

We regret to announce the death of Dr. Devereux Gwynne-Hughes, which occurred on February 8, 1948, at Sydney.

Nominations and Elections.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

MacFadzean, Reginald Victor, provisional registration, 1947 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

McKeon, Thelma Joyce, M.B., B.S., 1944 (Univ. Sydney), Canterbury District Hospital, Campsie.

Jordan, Audrey Ella, M.B., B.S., 1944 (Univ. Sydney), 52, West Parade, West Ryde.

Turner, Joyce Ingram, M.B., B.S., 1944 (Univ. Sydney), 45, Boundary Street, Roseville.

Tink, Arnold Richard, provisional registration, 1947 (Univ. Sydney), 39, Clifford Avenue, Manly.

McLachlan, Keith Roland, provisional registration, 1947 (Univ. Sydney), 104, Fitzroy Street, Grafton, New South Wales.

The undermentioned has applied for election as a member of the Victorian Branch of the British Medical Association:

Letham, John Burnside, M.B., B.S., 1946 (Univ. Sydney), Warburton, Victoria.

The undermentioned have applied for election as members of the Tasmanian Branch of the British Medical Association:

Oliver, Brian Houston, M.B., B.S., 1947 (Univ. Melbourne), General Hospital, Launceston, Tasmania.

Walpole, George Rex Outhwaite, M.B., B.S., 1946 (Univ. Melbourne), Devon Hospital, Latrobe, Tasmania.

The undermentioned have been elected as honorary associates of the New South Wales Branch of the British Medical Association:

Gray, Rex Justice, 5, Alison Road, Kensington.
Harris, Lelia, 51, Mooramie Avenue, Kensington.
Huber, Felix, 2/88, Drumalbyn Road, Bellevue Hill.
Johnson, Wilga, 43, Mary Street, Beecroft.
Rivett, Ronald Albert, 140, Edinburgh Road, Castlecrag.
Sander, Ralph, 6, Abbott Street, Coogee.
Schnitzler, George J., 77, Roslyn Gardens, Elizabeth Bay.
Tellesson, William George, 18, Athol Street, Coogee.

Notice.

INTERNATIONAL CONGRESS ON MENTAL HEALTH

An international congress on mental health will be held in London from August 11 to 21, 1948. Applications for membership should reach the congress organizer, 19, Manchester Street, London, W.1, before February 29. Application and hotel reservation forms may be obtained from Dr. W. S. Dawson, 135, Macquarie Street, Sydney.

Medical Appointments.

Dr. R. A. Burston has been appointed medical registrar at the Royal Adelaide Hospital, Adelaide.

Dr. J. H. Stace has been appointed anæsthetics registrar at the Royal Adelaide Hospital, Adelaide.

Dr. C. D. Jermyn has been appointed government medical officer at Kilcoy, Queensland.

Dr. David William Hawke has been appointed quarantine officer at Coff's Harbour, New South Wales, under the provisions of the *Quarantine Act*, 1908-1947.

The undermentioned appointments have been made at the Royal Melbourne Hospital, Melbourne: Physician to In-Patients, Dr. Clive Fitts; Physician to Out-Patients, Dr. W. E. King; Consulting Physician, Dr. W. W. S. Johnston; Consulting Orthopædist, Dr. C. W. B. Littlejohn.

Dr. G. E. Cole has been appointed a member and chairman of the Advisory Committee set up in pursuance of the provisions of Section 3 of the *Health (Patent Medicines) Act*, 1942, of Victoria.

Dr. H. B. Taylor and Dr. C. B. Cox have been appointed analysts, in pursuance of the provisions of Section 27 of the *Pure Food Act*, 1908, of New South Wales.

Books Received.

"Modern Audiometric Technique", by B. M. Green; 1947. Melbourne: Modern Hearing Aids Proprietary, Limited. 7¼" × 4¾", pp. 48. Price: 5s.

"Old Age: Its Compensations and Rewards", by A. L. Vischer, M.D., with a foreword by Lord Amulree, M.D., F.R.C.P.; 1947. London: George Allen and Unwin, Limited. 8½" × 5½", pp. 200, with illustrations. Price: 12s. 6d.

"Vade Mecum of Medical Treatment", by W. Gordon Sears, M.D. (London), M.R.C.P. (London); Fifth Edition; 1947. London: Edward Arnold and Company. 7" × 4¾", pp. 416. Price: 10s. 6d.

"Venereal Disease: Its Prevention and Conquest", by George Ryley Scott, F.Ph.S. (England), F.Z.S.; Second Edition; 1947. London. Torchstream Books. 7" × 4¾", pp. 80. Price: 3s. 6d.

"The 1947 Year Book of General Therapeutics", edited by Oscar W. Bethea, Ph.M., M.D., F.A.C.P.; 1947. Chicago: The Year Book Publishers Incorporated. 7" × 4¾", pp. 456, with illustrations. Price: $3.75.

"The Conduct of Life Assurance Examinations", by E. M. Brockbank, M.B.E., M.D. (Victoria), F.R.C.P.; The General Practice Series; Second Edition; 1947. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 176. Price: 12s. 6d.

"Practical Biology for Medical and Intermediate Students", by C. J. Wallis, M.A. (Cantab.); Second Edition Revised, Enlarged and Reset; 1947. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 406, with many illustrations. Price: 21s.

"Hearing Aids and Audiometers: Report of the Committee on Electro-Acoustics"; 1947. Medical Research Council of the Privy Council, Special Report Series Number 261. London: His Majesty's Stationery Office. 9½" × 6", pp. 76, with illustrations. Price: 1s. 3d.

Diary for the Month.

FEB. 21.—Tasmanian Branch, B.M.A.: Annual Meeting and Dinner.
FEB. 24.—New South Wales Branch, B.M.A.: Ethics Committee.
FEB. 25.—Victorian Branch, B.M.A.: Council Meeting.
FEB. 26.—South Australian Branch, B.M.A.: Branch Meeting.
FEB. 27.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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